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ARCHIVES OF OTOLARYNGOLOGY

VOLUME 15 JANUARY, 1932 NUMBER 1

THE OPERATIVE TREATMENT OF FACIAL PALSY

BY THE INTRODUCTION OF NERVE GRAFTS INTO THE FALLOPIAN CANAL AND BY OTHER INTRATEMPORAL METHODS *

SIR CHARLES BALLANCE, K.C.M.G., LL.D., F.R.C.S.
LONDON, ENGLAND
AND
ARTHUR B. DUEL, M.D.
NEW YORK

The History of the Operation of Nerve Anastomosis
The Early History of the Operative Treatment for Facial Palsy
The Choice of the Nerve to Be Grafted to the Facial Nerve:
1. The Hypoglossal Nerve
2. The Descendens Noni Nerve
3. The Glossopharyngeal Nerve
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Comments and Conclusions:
The Operation of Choice: The Introduction of a Nerve Graft into the Fallopian Canal

THE HISTORY OF THE OPERATION OF NERVE ANASTOMOSIS

The suture of a divided nerve, like many another established practice in modern surgery, seems to have been in use centuries ago. Guy

* Submitted for publication, April 24, 1931.

* The research on which this paper is founded was aided by the Carnegie Corporation, the Milbank Memorial Fund, the Lillian Babbitt Hyde Foundation and several personal friends.

de Chauliac,[1] for example, in a work compiled about 1363, after quoting from Avicenna and others the opinion that divided nerves should be sutured, and arguing that Galen did not, as some alleged, prohibit nerve suture, said: "I myself have seen and heard of many instances in which nerves and tendons have been divided, and have been so perfectly restored by suture and other aids that afterwards it seemed incredible that they had been divided."

Direct union of a divided nerve, though the best, is not the only operation available. It has been experimentally and clinically shown that restitution of function may be brought about by grafting the peripheral end of a divided nerve onto the central end of another nerve.

The earliest experiments on nerve anastomosis were those of Flourens.[2] He divided and crossed, in a cock, the two main nerves that go from the brachial plexus to the superior and inferior aspect of the wing. After some months, the function of the wing was reestablished.

It is desirable at the outset to state frankly that recovery after the union of one nerve with another, and even after direct suture of a divided nerve, though often wonderfully satisfactory as compared with the preceding paralysis, is never functionally perfect. What Sir James Paget[3] said of the effort of nature to return to the state that obtained prior to the application of a ligature to an artery in continuity is equally true of the healing of a divided nerve: "Gradually a nearer conformity of the tissue fabric, with the specific character of the parts it would replace, is established, till repair becomes almost reproduction."

THE EARLY HISTORY OF THE OPERATIVE TREATMENT FOR FACIAL PALSY

In 1879, "Drobnik united, in a man, the peripheral end of the facial nerve to the central end of the external branch of the spinal accessory. The palsy was due to suppuration in the petrous. After several months it was ascertained that the features had become more symmetrical."[4] We have not been able to find the original account of this case.

In 1895, one of us (Sir Charles Ballance[5]) united the facial nerve, end to side, to the spinal accessory. The facial paralysis had followed

1. de Chauliac, Guy: Chirurgica magna, tract III, doct. 1, cap. 3.

2. Flourens: Recherches expérimentales sur les propriétés et les fonctions du système nerveux dans les animaux vertébrés, ed. 2, Paris, J. B. Baillière, 1842. Ballance, C.: Anastomosis of Nerves, Arch. Neurol. & Psychiat. **25**:1 (Jan.) 1931.

3. Paget, J.: Surgical Pathology, London, Longmans, Green & Co., 1870.

4. Sawicki, Bronislas, in Chipault: L'état actuel de la chirurgie nerveuse, Paris, J. Rueff, 1902, vol. 2, p. 189.

5. Ballance, C.: Surgery of the Temporal Bone, New York, The Macmillan Company, 1920, vol. 2, p. 578.

an operation for mastoid disease. The result was that all the muscles on the palsied side of the face responded to faradic stimulation, but the patient never regained movement dissociated from the shoulder movement. No treatment of the muscles was carried out after the operation, as the man was lost sight of for five years.

In 1898, Faure [6] performed a spinal accessory facial anastomosis: The branch going to the trapezius was selected for the anastomosis. The palsy was caused by a bullet wound. When the case was reported, no power of voluntary movement had been regained. Following Faure's first case came the important experimental work of Barrago-Ciarella [7] and of Manasse.[8] These experiments were cited in detail by Bréavoine.[9]

The idea of anastomosing the facial to another nerve seems to have occurred independently to several surgeons: Gluck,[10] Kennedy,[11] Munch,[12] Girard [13] and Beck.[14] For example, Gluck, writing in 1908, said: "The idea that in facial palsy, an anastomosis should be done was conceived by me and published in 1896."

The nerve should be sought at the stylomastoid foramen and anastomosed to a neighboring nerve. The results obtained in man when the spinal accessory was united with the facial, whatever plan of anastomosis was adopted, were not satisfactory, in spite of some brilliant exceptions. Interference with the spinal accessory nerve was followed invariably by more or less deformity of the neck and shoulder, due to weakness or partial atrophy of the sternomastoid or trapezius, or both. This is shown clearly in a case described by Kummer [15] who performed the operation ascribed to W. W. Grant [16] of Denver. End-to-end anastomosis between the spinal accessory and the facial nerves was done, and then, in order to obviate the atrophy following the divisions of the spinal accessory, the distal cut end of this nerve was united end to end with the proximal cut end of the descendens noni.

Cushing,[17] in 1903, reported a case of spinal accessory-facial anastomosis. There followed drooping of the shoulder, and associated move-

6. Faure, quoted by Bréavoine (footnote 9).
7. Barrago-Ciarella: Policlinico (sez. chir.) **8**:124, 1901.
8. Manasse: Arch. f. klin. Chir. **62**:805, 1900.
9. Bréavoine: Trav. de neurol. chir., Paris, June 30, 1901.
10. Gluck: Deutsche med. Wchnschr. **28**:275, 1903.
11. Kennedy: Phil. Tr., Lond., s. B **202**:97, 1911.
12. Munch: Semaine méd. **24**:73, 1904.
13. Girard: Rev. méd. de la Suisse Rom. **26**:647, 1906.
14. Beck: Surgery of the Facial Nerve, Tr. Am. Laryng., Rhin. & Otol. Soc. **17**:265, 1908.
15. Kummer: Schweiz. med. Wchnschr. **50**:489, 1920.
16. Grant, W. W.: Traumatic Facial Paralysis, J. A. M. A. **55**:1438 (Oct. 22) 1910.
17. Cushing: Ann. Surg. **37**:641, 1903.

ments of the face when the shoulder was raised. There was, however, symmetrical closure of the eyelids without associated shoulder movement, and, after nine and one-half months, fairly symmetrical puckering of the lips occurred in the effort to whistle.

After spinal accessory-facial anastomosis, associated movements of the face and shoulder are always the first, and sometimes the permanent, result of the anastomosis. Dissociated movements of the face and shoulder are rare, requiring a long, patient period of reeducation, and may never be regained even after years of effort. A patient may, consciously or unconsciously, develop a habit of slightly contracting the muscles supplied by the spinal accessory nerve when a movement of the muscles of the face is attempted. Again, symmetrical, subconscious, emotional movement of both sides of the face—in which the cortical centers of opposite sides of the brain must cooperate in unison—the movement that is most desired, is the one that most frequently eludes recovery.

THE CHOICE OF THE NERVE TO BE GRAFTED TO THE FACIAL NERVE

It is not surprising, therefore, that several surgeons, noting the imperfections in facial control following the use of the spinal accessory nerve, should have made a choice of some other nerve for the anastomosis, in the hope that better results would be attained. The choice lay between the hypoglossal, the descendens noni and the glossopharyngeal nerves.

1. *The Hypoglossal Nerve.*—Furet [18] suggested to Faure that facial palsy might be cured by anastomosing the facial with the hypoglossal nerve. Faure, however, performed the operation in 1898, employing the spinal accessory nerve in preference to the hypoglossal.

In April, 1903, at the German Surgical Congress in Berlin, Professor Korte [19] described a case in which he had performed a hypoglossal-facial anastomosis. One of us (Sir Charles Ballance) was present, and described a similar case in which the patient had been operated on at a somewhat later date.

In May, 1903, Bernhardt,[20] professor of neurology in the University of Berlin, wrote as follows about Korte's case:

> The patient in laughing moves the mouth, and movements of expression can also be carried out through the facial. I believe, however, there are associated movements of the tongue which escape observation, as the tongue is confined within the mouth, but they in no way trouble the patient, nor are they noted by those

18. Furet, quoted by Bréavoine (footnote 9).
19. Korte: Deutsche med. Wchnschr., 1903, no. 17.
20. Bernhardt: Private letter to Sir Charles Ballance.

around her. From all that I have observed, I entirely agree with you that in grafting the facial for the relief of facial palsy, it is better, notwithstanding the opinion of Korte, to choose the hypoglossal rather than the spinal accessory. I have had a recent opportunity of confirming the opinion, as Professor Gluck, at the Berlin Society for Psychiatry and Nervous Diseases, showed a boy on whom he had grafted the peripheral end of the paralysed facial on to the spinal accessory. Here only associated movements of the facial muscles were present. As in Kennedy's case, it was only when the boy raised his arm that the muscles of the face contracted; but in neither Gluck's nor Kennedy's case could movements of expression be brought about.

Professor Tilmann's [21] case of hypoglossal-facial end-to-end anastomosis is an excellent example of successful operation. The final result

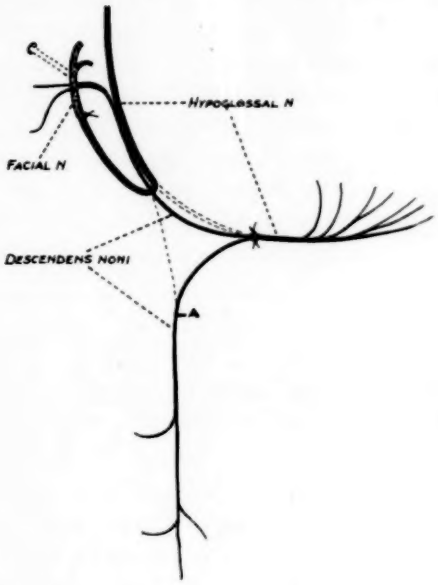

Fig. 1.—Hypoglossal-facial end-to-end anastomosis; hypoglossal-descendens noni end-to-end anastomosis.

four years after operation (August, 1904) was due to his long and persistent efforts to reeducate the facial muscles.

After nine months it was observed that the left side of the face moved slightly with the movements of the tongue. These movements, at first very slight, gradually grew stronger, until finally, strong movements of the face occurred every time the patient moved his tongue. The chief difficulty, however, is to reproduce the mimetic movements of the face, for instance, in reading. Whenever the patient swallows, there is always a very slight associated movement (after four years). When the patient puts out his tongue, the left half is seen to be atrophied, but the patient makes no complaint of any sort. The absence of the left hypoglossal causes

21. Tilmann: Verhandl. d. deutsch. Gesellsch. f. Chir., 1909.

no essential disturbance, but the absence of the spinal accessory produces severe cosmetic disturbances: The shoulder sinks, is directed more forward, and the outline of the neck is distorted.

When one of us (Sir Charles Ballance) was in London last Christmas, he saw a woman on whom, nine years before, he had performed a hypoglossal-facial anastomosis. On fixation of the tongue and swallowing, a slight ripple of muscular contraction passed over the side of the face.

In the reported cases of Lafite-Dupont,[22] Bevers [23] and others, an end-to-side hypoglossal-facial anastomosis was carried out. We have performed end-to-side hypoglossal-facial anastomosis, and the experience has persuaded us that it is a mistake to attempt the great problem of curing facial palsy and, at the same time, to endeavor to preserve the function of the tongue unimpaired. We think, too, objectives in surgery, as in war, mean, as a rule, partial success or failure. The curing of facial palsy justifies the sacrifice of the movements of half of the tongue. The end-to-side anastomosis may militate against a more perfect restoration of the facial movement. The only operation, therefore, that should be carried out is end-to-end anastomosis. "On this earth of ours what great good has ever been or can be gained except by sacrifice? It is, in every era, and in every zone, the law of life." [24]

2. *The Descendens Noni Nerve.*—The operation of descendens nonifacial end-to-end anastomosis has been done in man. Several experiments of the same type have been done on baboons and rhesus monkeys. In addition, the distal severed end of the descendens noni is united end-to-side to the lower border of the hypoglossal nerve. This second anastomosis prevents permanent paralysis and atrophy of the depressor muscles of the hyoid bone and larynx. Thus, when the descendens noni is employed in this anastomosis, there is no permanent atrophy of muscle, as there is when the spinal accessory or hypoglossal nerve is used. In the monkey, however, there are remarkable associated movements. In swallowing, when the depressor muscles contract, the eyelids are in constant movement, frequently closing over the eye-ball, and further rapid to and fro movements of the pinna occur as if the retrahens aurem muscle was in intermittent contraction.[25]

3. *The Glossopharyngeal Nerve.*—In a letter to one of us (Sir Charles Ballance) in 1895, Professor Schafer,[26] then professor of physiology at University College, London, suggested the glossopharyngeal as a suit-

22. Lafite-Dupont: Congrès Soc. franç. d'otol., 1906.
23. Bevers: Lancet **1**:1450, 1913.
24. Roberts: Address, The Times, March 15, 1913.
25. Ballance: Brit. J. Surg. **11**:341, 1923.
26. Now regius professor of physiology in the University of Edinburgh.

able nerve to attach to the divided facial nerve, on the ground that its motor nucleus is near to, and apparently serially homologous with the facial nucleus. The advice was not taken; the spinal accessory and later the hypoglossal were used, because these nerves were larger and, it was thought, more accessible. Such arguments are futile and stultifying when employed in opposition to the best scientific opinion.

In man, the glossopharyngeal nerve has been united by one of us (Sir Charles Ballance) to the facial, five times: Once [27] the operation, in a long-standing case of facial palsy, was a typical end-to-end junction of the facial and glossopharyngeal nerves, and in four instances the operation consisted of end-to-end anastomosis of the facial to both the glossopharyngeal and descendens noni nerves. One of the patients was under the care of the late Sir William Milligan. The child was 7¾ years old. At the age of 4 months, she had suffered from tuberculous mastoiditis which destroyed the facial nerve. One year after the nerve anastomosis operation was done, faradic contractility and voluntary movement had returned in the previously paralyzed face, but voluntary movement was as yet weak at the angle of the mouth, as in laughing the muscles of the opposite side of the face had a tendency to pull the mouth over to that side. The child had had no treatment of the paralyzed muscles for seven years, and possibly some of the fibers of the paralyzed muscles had disappeared.

The reinforcing of the glossopharyngeal by the descendens noni was carried out in consequence of doubt arising as to the value of the early glossopharyngeal-facial experiments performed in monkeys, care not having been taken to exclude the possibility of union between the divided ends of the facial nerve.[28] At the same time, the employment of two nerves made it impossible to decide what part, if any, each nerve took in the evolution of the recovery of the facial paralysis. Watson-Williams [29] reported two cases of glossopharyngeal-facial anastomosis. In one of these cases the facial nerve was divided during the performance of a labyrinthectomy. Seven months later the facial nerve was anastomosed to the glossopharyngeal nerve. Eighteen months after the nerve anastomosis, the facial palsy had almost disappeared. In the other case facial palsy followed an operation for mastoid disease in 1916. In 19?? a glossopharyngeal-facial anastomosis was done. Fifteen months after the nerve anastomosis, the patient could whistle.

In the patient operated on by one of us (Sir Charles Ballance) it was noted that the division of the glossopharyngeal nerve caused no difficulty in swallowing, and that there was no indication that the pro-

27. Ballance: Brit. M. J. **2**:349 (Aug. 30) 1924.
28. Ballance (footnote 2, reference 2, fig. 10).
29. Watson-Williams: J. Laryng. & Otol. **42**:516, 1927.

tection of the larynx against the entrance of food was impaired. Further, five months after division of the glosso-pharyngeal, common sensation and taste appreciation of the back of the tongue on the side of the operation had returned; indeed, no difference could be detected on the right and left side of the tongue.

We know, then, that following glossopharyngeal-facial anastomosis there is no serious physical defect or loss of sensation of the tongue. In recent experiments on the baboon satisfactory results have been obtained in regard to the recovery of the muscles of the face. But in all the baboon experiments more or less associated movements were observed. In one baboon, three and one-half months after operation, during eating and swallowing, the eyelids on the side of the operation were seen nearly to close, and there was some movement of the upper lip. How long these movements last we do not know; neither do we know if they are permanent, like those already described when the hypoglossal nerve is anastomosed to the facial nerve. Our impression is that they become less as time goes on, but we are ignorant as to whether they ever disappear altogether.

Finally, if the facial nerve is to be anastomosed to another nerve, the baboon experiments point to the glossopharyngeal as the nerve of choice to employ. The best recoveries following glossopharyngeal-facial anastomosis in baboons certainly appear to outclass the recovery from hypoglossal-facial anastomosis. We do not propose, however, to offer advice on this question, our object in this paper being to urge the performance of the operation that brings again into union the two ends of the divided facial nerve.

STAGES IN RECOVERY FROM FACIAL PALSY AND THE REASSUMPTION OF CONTROL OF THE FACIAL MUSCLES BY THE NERVE CELLS OF THE ROLANDIC CORTEX

Frazier and Spiller [30] reported a case of hypoglossal-facial end-to-end anastomosis in 1903, and added a thoughtful commentary.

Frazier described three stages of improvement leading to recovery: (1) the restoration of normal tonus, so that at rest the face is symmetrical; (2) the stage in which voluntary movements are possible, and (3) the stage of attainment of symmetrical movements induced by emotional stimuli.

Both Frazier and Spiller recommended the use of the whole nerve instead of only a part: "The disadvantages are minor when the overcoming of the severe deformity caused by facial palsy is under consideration." There was no mention of associated movements in the

30. Frazier and Spiller: Univ. Penn. M. Bull. **16**:306 (Nov.) 1903.

report of this case. This important point was also omitted in the records of other cases. Spiller wrote:

> The return of function need cause no surprise if we remember that when a motor nerve is divided, and at once reunited, there is not a reunion of the same fibres, but different fibres unite so that other nerve cells are brought into association with the peripheral ends of the divided nerve fibres. If this can occur between the nerve fibres of the same bundle, there seems to be no reason why it may not occur between the nerve fibres of different bundles.

The fact is that, as other neurologists have affirmed, there is no fundamental difference between the nerve fibers. The function of the peripheral nerve depends on the cells of the cerebral center and of the nerve ending being intact.

The question of how the brain cortex reassumes control of the facial muscles is best understood by the study of other cases in which the conditions present are somewhat comparable to facial paralysis after nerve anastomosis has been done. Spiller wrote:

> When the speech centres of the left side of the brain are destroyed in early life the power of speech usually returns. We assume that the dormant centres on the right side of the brain acquire the function that would otherwise belong to the centres of the left cerebral cortex. Here is an illustration of the education of a new centre in the brain for the performance of a function after the usual centre is destroyed.

Again Spiller described the brain of a boy who was able to walk although the motor cortex and motor tract on one side had been destroyed; the anterior pyramid of the opposite side was larger than normal. In this case, volitional movements of the limbs of both sides of the body were controlled by the centers of one cerebral hemisphere. It is clear that the substitution of one area of the cortex for a similar one is possible, especially in the young. But the question presses: Are these cases exactly comparable to the case of an anastomosis of the facial nerve with another nerve? Do the cells of the center in the rolandic cortex, which controls the movements of the muscles of the face, atrophy and become no longer of any service in the case of facial paralysis, say, of six months' standing? We do not think so, but only experiment can decide. No experimenter has as yet divided the facial nerve in the baboon, allowed the muscles of the face on the side of operation to remain completely paralyzed for six months, then grafted the severed distal end of the facial onto some other nerve and, lastly, when faradic excitability and voluntary movement have returned in the muscles, applied an electrical stimulus to the appropriate motor area of the opposite cerebral cortex.

When the paralysis of the face has lasted, say, for six or nine months, the paralyzed side of the face is flattened, and it is difficult to

palpate the muscles. Nevertheless, the severance of the facial nerve at the stylomastoid foramen, in order to perform an anastomosis, will be noticed the next day, on careful examination, to have caused the face to appear more flattened and more paralyzed than before the operation. The nerve has in part regenerated after degeneration, and we suggest that the chain of cells in the length of the nerve acts like a row of cells of an electric battery. An impulse of chemical or of some other nature is carried to the paralyzed muscles. This influence is interrupted by the division of the facial nerve at operation.

Again, when the facial nerve has been anastomosed to another nerve for the cure of facial palsy, long before a nervous impulse passes that is competent to induce voluntary motion or faradic response, certain remarkable changes in the muscles occur. The appearance of the face, the added plumpness of the muscles and the statement of the patient that he feels something, difficult to describe, creeping about the face are the early indications of recovery. These signs, which commence very early, certainly a month after an anastomosis and probably earlier, show that an impulse is passing the anastomosis that has for its function the chemical regeneration of the paralyzed muscles. Anyway, long before the function of the muscles is completely restored, something is happening along the course of the nerve that is vitally important to the rehabilitation of the muscles and that is not at present understood. These changes in the muscle fibers would seem to be associated with the static motility so clearly described by Ramsay Hunt. The same events in regard to the recovery of the muscles of the face have been observed in the anastomosis experiments in baboons, in which the living facial nerve was divided and healthy muscles were suddenly paralyzed. If this is so with regard to the paralyzed muscles of the face, might not some such influence antagonize retrograde changes in the cells of the rolandic cortex? The problem of the recovery of the muscle cells and nerve cells opens an interesting field for further investigation.

The observations described contribute support to the thesis that there is a dual innervation of striated muscle. Of this there can be hardly any doubt. In a philosophic study entitled "The Dual Nature of the Efferent Nervous System" Ramsay Hunt [31] wrote:

> The function of motility may be resolved into two components. . . . One of these is the movement proper, which is subserved by the kinetic system. The other component is the more passive form of contractility underlying postural tone, and is subserved by the static system. The term static is used here to designate that peculiar property of muscle by which it becomes fixed in posture. . . . The striated muscle fiber contains a motor nerve ending which is the terminal of a medullated nerve fiber, and another nerve ending—the terminal of a nonmedullated nerve. . . . The striated muscle fiber is composed of two substances: one sub-

31. Hunt, J. Ramsay: The Dual Nature of the Efferent Nervous System, Arch. Neurol. & Psychiat. **10**:37 (July) 1923.

serving a contractile and the other a postural function. . . . A striated muscle fiber consists of two distinct types of contractile substance which present distinct forms of chemical activity which are probably related to the two forms of activity: the contractile and postural functions.

EXPERIMENTAL INVESTIGATIONS

A. Experiments Since the Great War.—After the war, and before one of us (Sir Charles Ballance) came to America, thirty experiments were performed. The trunk of the facial nerve, divided at the stylomastoid foramen, was anastomosed to the (1) hypoglossal nerve, (2) descendens noni nerve, (3) descendens noni and communicans noni nerve, (4) masseteric nerve, (5) lingual and chorda tympani nerve, (6) chorda tympani nerve, (7) glossopharyngeal nerve and the (8) cervical sympathetic nerve.

It may be stated at once that whatever nerve of the neck was grafted onto the facial nerve, faradic contractility returned in the muscles of the paralyzed face, except in one instance in which the wound was torn open and the anastomosis destroyed. At the end of six months the amount of recovery of the face varied; e. g., in the lingual-facial anastomosis, a strong faradic current was required to elicit good muscular contraction. It would seem, indeed, that as long as the nerve cells of the activating nerve center and the motor end-organs in the muscles are intact, it does not matter much which pathway is chosen for carrying the nerve impulses between them; in other words, when the usual road is blocked, another pathway is available.

Of the aforementioned experiments, ten were cervical sympathetic-facial anastomoses and one a sympathetic-hypoglossal anastomosis. The records of these have been reported elsewhere. In nine of the experiments, the glossopharyngeal was united end to end with the facial nerve. The recovery of the face in the experiments on monkeys appeared to be the most satisfactory in those in which the glossopharyngeal nerve had been employed. In three months, all the muscles responded to the faradic current; the face at rest was symmetrical, but the eyelids did not as yet wink together synchronously. At the end of six months, voluntary movement and faradic response in the muscles of the previously paralyzed faces were normal in several of the animals; at any rate, scarcely the slightest difference could be detected in comparing the movements and the electrical response of the muscles of the two sides of the face. No external physical defect could be detected, although associated movements, varying from a slight partial closure of the eye to movements involving other parts of the face and pinna, were present.

The glossopharyngeal nerve consists of a small motor element, which supplies the stylopharyngeus muscle, and a larger sensory element. The lingual branches supply the vallate papillae and the mucous membrane

of the dorsal third and lateral half of the tongue, extending backward to the glosso-epiglottic folds and the front of the epiglottis. The motor root of the glossopharyngeal takes its origin from the nerve cells at the upper part of the nucleus ambiguus. From the rest of the cells of the nucleus ambiguus, the motor fibers of the vagus and the motor fibers of the bulbar part of the spinal accessory arise. Above the nucleus ambiguus is the motor root of the facial nerve, and below it is the anterior horn of gray matter of the spinal cord. From the motor cells of the outer part of the horn arise the fibers of the spinal portion of the spinal accessory nerve. The close proximity of the motor nuclei of the facial, glossopharyngeal and spinal accessory nerves, also the juxtaposition of the petrous ganglion of the glossopharyngeal to the ganglion of the trunk of the vagus, which receives branches of communication from the spinal accessory nerve, seemed to make it advisable to determine if any motor fibers of the spinal accessory or facial nerves reached the glossopharyngeal nerve. No degeneration was observed (Marchi method) in the glossopharyngeal:

1. After division of the spinal accessory within the skull.

2. After division of the chorda tympani in the tympanum.

3. After division of the facial nerve at the stylomastoid foramen. This section also divides the ansa of Haller (the communication [32] between the facial and glossopharyngeal nerves outside the skull).

4. Testut also figures a "rameau lingual de facial" (his fig. 85), which comes from the facial nerve outside the skull and extends to the back of the tongue. It communicates by two branches with the glossopharyngeal nerve. This nerve must have been severed when the facial nerve was sectioned outside the stylomastoid foramen. No degeneration was seen in the glossopharyngeal nerve.

5. The small superficial petrosal nerve communicates by a minute filament with the tympanic plexus and Jacobson's nerve, but its influence on the glossopharyngeal nerve may be ignored.

The conclusion, therefore, is that the recovery in cases of glossopharyngeal anastomosis is due directly to the action of the cells of the motor nucleus of that nerve.

Toward the close of these experiments, a cervical-sympathetic-facial anastomosis was done in a baboon. The main trunk of the facial nerve was very short, about one-eighth inch (0.3 cm.) in length. The question arose whether the nearness of the anastomosis to the stump of the facial would allow of union between the severed ends of the facial nerve. Never before had so short a facial trunk been seen. The distance between the divided facial nerve at the stylomastoid foramen and

32. Testut: Traité d'anatomie humaine, ed. 5, Paris, O. Doin, vol. 3, fig. 84.

the anastomosis was disquietingly short. As a rule, the facial trunk is a good half inch (1.2 cm.) in length, and when it is turned forward, sometimes after the posterior auricular branch is severed, the site of the anastomosis is an inch (2.5 cm.) away from the stylomastoid foramen. Two monkeys were still alive, besides the one mentioned, in which a glossopharyngeal-facial anastomosis had been done. The brain of each was removed, and the facial and glossopharyngeal nerves were stimulated with the faradic current in the posterior fossa of the base of the skull. In one experiment, contraction of the facial muscles of about equal strength occurred, with stimulation of the facial and the glossopharyngeal nerves. In the other experiment contraction of the muscles took place when the glossopharyngeal was stimulated, but no response in the muscles occurred when the facial was stimulated. The results threw the conclusions that had been arrived at, in regard to each one of these experiments, into the melting pot. In man, the facial nerve at the stylomastoid foramen after long-standing palsy is in a different state from that which obtains in the facial nerve of an experimental animal. In man, the facial nerve is alive, it is true, but it has degenerated and then in part regenerated; it is not in the actively living state of the facial nerve when divided in an experimental animal. In man, recovery is the main quest, and it matters not how that recovery takes place. The damaged nerve lies within the temporal bone, and the part of it distal to the injury is incapable of assisting in the recovery of the motor function of the face muscles; otherwise an anastomosis with another nerve would not have been necessary. In the monkey, in which a scheme of research has been planned in order to find out which nerve of the neck it is best to use for anastomosis with the facial, it is imperative that the experiment should be so conceived and carried out that the recovery of the face could not be due to the interference of any other nerve than that one chosen for anastomosis with the facial nerve.

B. Experiments Carried Out in the Laboratory at Laurelwood Since July, 1930.—1. The trunk of the facial nerve was anastomosed to: (*a*) the glossopharyngeal nerve (six experiments on baboons), (*b*) the hypoglossal nerve, (*c*) the descendens noni nerve, (*d*) the lingual nerve, (*e*) the superior laryngeal nerve and (*f*) the auriculotemporal nerve.

2. At a later date in rhesus monkeys and cats, the facial nerve was grafted to: (*a*) the great auricular nerve, (*b*) the great occipital nerve, (*c*) the transverse cervical nerve, (*d*) the auriculotemporal nerve and (*e*) other sensory and motor nerves.

3. Lastly, certain intratemporal operations were performed on baboons, rhesus monkeys and cats.

It will be noted, on referring to the protocols of the first series of seventeen experiments, that in all the anastomosis experiments, the facial nerve was removed from nearly the whole length of the fallopian canal. These experiments, however, are not yet complete. We propose to stimulate the cortex of each monkey so as to see whether a response can be obtained in the muscles of the face by nerve impulses traveling along the nerve grafted onto the facial; and then, after the brain is removed, to stimulate intracranially the facial nerve and the anastomosing nerve. It cannot be imagined, we think, that after the facial nerve has been ablated from the fallopian canal, it can in any way vitiate the results of these experiments. It is imperative that no doubt should arise in any mind as to the justification of our conclusions.

The anastomosis experiments show: (*a*) The comparatively satisfactory result of glossopharyngeal-facial anastomosis. (*b*) The remarkable result of auriculotemporal-facial anastomosis, considering that the auriculotemporal is a comparatively small sensory nerve.

The intratemporal experiments show: (*a*) The result of division of the facial nerve in the fallopian canal, and the value of decompression. Decompression of the facial nerve in the fallopian canal is not a new method, but its paramount importance in the healing process of the facial nerve has not been generally recognized. (*b*) The success attending the introduction of a nerve graft into the fallopian canal.

The experiments exhibit a new method of operation for the cure of facial palsy which, we believe, holds out greater promise of more nearly perfect recovery than any plan yet devised.

THE HISTORY OF INTRATEMPORAL OPERATIONS IN MAN

1. *Decompression of the Facial Nerve.*—When facial palsy occurs in the course of acute or chronic otitis, the appropriate operation on the mastoid should be done immediately. Alt [33] recommended that in such cases careful search should be made, at the conclusion of the mastoid operation, for any erosion of the wall of the fallopian canal and that the canal should be deliberately opened and the nerve inspected and freed by careful wiping away of granulations and fragments of cholesteatoma. In this way, all pressure is removed, and rapid recovery is the rule. He related an instance in which, on operating for cholesteatoma in a woman aged 32, who had complete facial palsy before the operation, he found an opening 3 mm. long in the facial canal. He exposed the nerve for some distance above and below this, turned it out of the canal, cleansed it by wiping and replaced it in the groove.

33. Alt, F.: Verhandl. d. deutsch. otol. Gesellsch., 1908, p. 191.

In seven months recovery was complete. Alt's operation, performed in 1908, is a model for all to reflect on, and a typical illustration of the value of decompression.

In facial palsy from injuries, such as incised wounds, stabs, gunshot wounds and fractured bone, spontaneous recovery is improbable, while in facial palsy, so-called rheumatic palsy, due to inflammation of the geniculate ganglion, there is a tendency toward recovery. Kummel[34] expressed the opinion that when facial palsy was caused by a chisel stroke, either an angular fracture of the wall of the canal occurred or a fragment of the wall was driven between the two ends of the nerve at the moment of division. He advised that the nerve should be exposed above and below the site of injury, its ends freshened and the intervening bone removed. This recommendation is another example of how to deal with facial palsy by decompression. Decompression is the obvious course to pursue.

When the palsy is due to compression by scar tissue in the tympanum and in the injured canal, the immediate method to adopt is to remove this tissue, just as in some cases of musculospiral paralysis a cure is effected by removing callus and scar tissue compressing and damaging the nerve.

Kennedy[11] succeeded in obtaining partial recovery from facial palsy that had lasted fifteen years. Before operation, however, the muscles responded to the faradic current. The injury had been caused by a mastoid operation when the girl was 10 months old. The inner wall of the tympanum was covered by a thick layer of cicatricial tissue and granulations. This was dissected away, and at the bend of the canal a soft and yielding patch was found. The nerve was then dissected out, and the external wall of the aqueduct chipped away so as freely to liberate the nerve from pressure. The nerve was then lifted out of the canal. It looked like a nerve that had been divided and reunited satisfactorily. Improvement was slow, and was not complete when the report was made. The question arises: Would it have been better to cut out the damaged portion of the nerve and to have filled the interval with a nerve graft?

2. *Direct Suture or Apposition of the Ends of the Divided Nerve Without Displacement from the Canal.*—This is the ideal surgical procedure, and should be carried out, whenever practicable, in cases of division of the nerve, whether recent or of old standing.

Stacke,[35] in 1903, reported a case in which direct union of the divided nerve was brought about by operation. Facial palsy had been

34. Kummel: Verhandl. d. 74 Versammlung d. Gesellsch. d. Kinderh. deutsch. Naturf. u. Aerzte.

35. Stacke, quoted by Alt (footnote 33).

caused by a mastoid operation. The nerve had been injured at the bend and was embedded in old firm granulation tissue. The nerve was divided, turned out of the canal and freed from scar tissue, and the two ends were replaced in position.

A similar procedure was carried out by Sydenham,[36] who used a piece of silkworm gut laid between the two ends of the nerve in the aqueduct. The operation was done on the second day after the division of the nerve. The first sign of returning function was seen at the end of three months.

Marsh[37] reported two cases (1909) in which he had adopted a similar treatment, one in 1900 and one in 1908. In the former patient, operated on two weeks after division of the nerve, a piece of catgut was inserted into the aqueduct between the severed ends of the nerves. Complete recovery resulted. In the second case, similar measures were adopted one month after the injury which also occurred during the mastoid operation. A partial recovery took place, but was complete in the lower part of the face.

3. *Displacement of the Nerve from the Canal; Removal of Damaged Portion and Suture.*—The aqueduct forms a curve around the posterior and upper boundaries of the tympanum proprium. The nerve can be lifted out of the canal, the injured part cut away, and the ends of the nerve then sutured in the region of the promontory. The shortened route from the region of the geniculate ganglion allows the divided ends to be brought into contact without tension. A good 3 mm. is gained by this shortened route. One of us (Sir Charles Ballance) performed the operation in 1894, but it was not successful, probably in consequence of sepsis. R. C. Martin[38] recently reported an interesting case in which this plan was adopted. The nerve was freed from the stylomastoid foramen, which allowed that part of it to be coaxed backward and upward, thus allowing the displaced ends of the nerve to be brought together without tension. When the case was reported, recovery from the paralysis was proceeding satisfactorily. Martin recommended the operation in cases of Bell's palsy, but, surely, in Bell's palsy only decompression would be necessary.

COMMENTS AND CONCLUSIONS

1. Anastomosis Operations in Which the Facial Nerve is United to Another Nerve
2. Nerve Graft Operations Designed to Assist in the Union of the Two Ends of the Divided Facial Nerve

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36. Sydenham: Brit. M. J. **1**:1113, 1909.
37. Marsh: Brit. M. J. **1**:1356, 1909.
38. Martin, R. C.: Intratemporal Suture of Facial Nerve, Arch. Otolaryng. **13**:259 (Feb.) 1931.

3. The Operation: Preparation by the Surgeon:
   (*a*) First Stage: the Mastoid Operation
   (*b*) Second Stage: the Operation on the Aqueduct of Fallopius
   (*c*) Third Stage: the Introduction of a Nerve Graft
   (*d*) Fourth Stage: the Dressing and Aftertreatment
   (*e*) Case of facial palsy
4. When Is It Justifiable to Operate?
5. The Choice of the Time to Operate
6. Is Suppuration a Reason for the Postponement of the Operation on the Nerve?
7. The Causes of Facial Palsy:
   (*a*) Mastoid Disease and Mastoid Operations
   (*b*) Bell's Palsy:
      1. Geniculate Ganglion Inflammation
      2. Inflammation of the Nerve in the Lower Half of the Descending Portion of the Canal
   (*c*) Fracture of the Middle Fossa of the Base of the Skull; Gunshot Wounds and Stab Wounds; Tumors

1. *Anastomosis Operations in Which the Facial Nerve Is United to Another Nerve.*—Reflecting on the early operations for facial paralysis, and the delight experienced in seeing the paralyzed muscles of the face in movement again, the surgeon (and we speak for ourselves) allowed the splendor of the recovery to conspire to blind his erring judgment and to misguide his mind. There can be at the present time no question as to the disability attending an anastomosis operation, and as to the imperfections present in many cases that were hailed as good recoveries in the past. In further considering the future of the operation for the relief and cure of facial palsy, we are driven to the conclusion that the anastomosis operations of the past may be looked on correctly now as a stage in the evolution of this branch of surgery. In thus expressing our considered opinion, we pass on from thirty-five years of operations on patients and of experimental investigations by many workers to a clearer vision of the problem to be solved and, we also believe, to a brighter promise for the practice of surgery in the treatment for facial palsy. One of the fundamental principles of surgery, which allows of few exceptions, is that a bleeding artery must be dealt with at the site of hemorrhage. It is also a fundamental principle of surgery that an injured nerve must be operated on at the site of injury. The surgeon seeks progress and perfection in his methods, but his results rarely attain to the standard of perfection—

> Whoever thinks a faultless piece to see,
> Thinks what ne'er was, nor is, nor e'er shall be.[39]

39. Pope: Essay on Criticism, line 253.

After all, it is an unnatural thing to anastomose one nerve to another, when the ideal surgical method of bringing the two ends of a divided nerve into contact, either directly or by means of a nerve graft, is both feasible and practicable. Pope's lines are a reenforcement of the argument:

> First follow *Nature*, and your judgment frame
> By her just standard, which is still the same;
> Unerring *Nature*, still divinely bright,
> One clear, unchang'd and universal light,
> Life, force and beauty must to all impart,
> At once the source, the end, the test of *Art*.[40]

If the facial nerve had had its home among the soft tissues, the surgeon would never have practiced anastomosis operations. There appears no cogent reason, because the nerve lies in a bony tube, that an anastomosis operation should be done. On the contrary, the operator's duty is to treat the injury at the site of injury, and not struggle to obtain recovery by the indirect and roundabout way of anastomosis. By exposing and dealing directly with the damaged portion of the nerve, the surgeon's effort will result in a better recovery; no associated movement or paralysis of muscles subserving other regions will mar recovery. We hold strongly that anastomosis operations for the cure of facial paralysis should be banished from future surgical practice. Nevertheless, to every law there are exceptions. When the facial nerve in all its long course within the fallopian canal has been destroyed, for example, by necrosis of the petrous or by gunshot wound, so that an intratemporal nerve-grafting operation is not possible, there is nothing left to do but to perform the old operation of anastomosis.

2. Nerve Graft Operations Designed to Assist in the Union of the Two Ends of the Divided Facial Nerve.—Létiévant,[41] in 1873, recommended nerve grafting as a possible expedient when the ends of a divided nerve could not be brought together, and added that it had been often successfully done in animals.

Purves Stewart and one of us (Sir Charles Ballance),[42] in 1891, reported several cases in man, in which the sciatic nerve of the sheep, or of the bullock, was used to fill gaps in the great nerves caused by gunshot wounds. If nerve grafting is the operation decided on, the graft should by preference be autoplastic and not heteroplastic; but when a portion of a large nerve has been shot away, it is only when an amputation in another patient becomes necessary that it is possible

40. Pope (footnote 39, line 68).

41. Létiévant: Traité du section nerveuse, Paris, J. B. Baillière et fils, 1873, p. 44.

42. Ballance, C., and Stewart, Purves: The Healing of Nerves, New York, The Macmillan Company, 1901.

to obtain a living autoplastic nerve graft for the purpose. When it is desirable to introduce a nerve graft into the fallopian canal, no difficulty arises in obtaining an autoplastic graft.

Sick and Sänger,[43] in 1893, in a case of divided musculospiral nerve joined the ends together by a bridge formed of half the thickness of the median nerve. Sixteen months later, the extensor palsy had gone, with the exception that the thumb could not be extended.

We did not arrive at the decision to employ a nerve graft in the fallopian canal by a sudden inspiration, like the sudden flaming light of a tropical dawn. The method was conceived slowly after nearly a year of experimentation on baboons, rhesus monkeys, cats and Louisiana frogs, in which 130 experiments were performed. Some of the experiments were not directly associated with the problem of the cure of facial palsy. Many of them were related to the question of the conduct and fate of nerve grafts. Many are not yet complete, as the final picture of recovery, after the division and suture of a nerve, with or without the use of a nerve graft, is not available until six or more months have elapsed. From all the experiments there was something of value to learn concerning the surgical argument elaborated in the paper. The natural sciences are so closely linked together that the investigation of one sheds light on all the others. We have in view further contributions in the near future.

As experiments matured, the sunrise of a new and wonderful plan of treating patients for facial palsy was gradually unfolded before our vision. At first, we took various lengths of the facial nerve out of the facial canal, reversed them and replaced them within the canal. Then, we divided the facial nerve in the canal, and, lastly, removed portions of the facial nerve from the fallopian canal and substituted similar lengths of some other nerve, such as the intercostal nerve, the descendens noni nerve or the external respiratory nerve of Bell. The uniform and remarkable success that attended these experiments appears to offer proof that this new plan of operation is better than any other for the cure of facial palsy in man.

3. *The Operation: Preparation by the Surgeon.*—The operation cannot be done casually by the light of Heaven. It demands preparation and the careful thought of the surgeon. The operation is a delicate one, and to win success the essentials are as follows: (*a*) In the first place, the surgeon must have a clear idea in his mind of the structures among which he has to find his way to the nerve. "'I always determine in my own mind,' said the sculptor Chantrey, 'the expression to be given, and, unless I can see the face distinctly and with that

43. Sick and Sänger: Arch. f. klin. Chir. **55**:271, 1897.

expression when I close my eyes, I can do nothing.'"[44] (*b*) There must be brilliant illumination of the field. (*c*) The surgeon must possess skilled manipulative ability.

Before describing the operation on the fallopian aqueduct, it is well to repeat that the operation is by no means easy to perform. Just as well surveyed waters, though narrow and tortuous, may be safely and confidently navigated, so the surgeon learned in anatomy, and with the knowledge and skill gained in the deadhouse, may safely traverse "the perilous narrow ocean"[45] of the operation on the fallopian aqueduct.

There is, indeed, only a narrow way going up to the top of the "Hill Difficulty," and it is in that narrow way that the surgeon's feet should tread. On either side of the narrow way are two other ways, and the name of one is "Danger" (the external semicircular canal) and the name of the other is "Destruction" (the oval window). "Now when Christian was got up to the top of the hill, there came two men pressing against him amain; the name of the one was Timorous and the name of the other was Mistrust. To whom Christian said, 'Sirs, what's the matter you run the wrong way?' Timorous answered: 'The further we go, the more danger we meet with, wherefore we turned and are going back again.'"

The surgery of the temporal bone is, as Greig-Smith[46] so well said of another department of surgery, "no longer a field for legitimate and versatile experiment; certain fixed and useful laws and customs have been laid down by the dearly bought experience of great men; the Surgeon ought to begin fully equipped with such knowledge as has been gathered for him."

The spirit of Christian in climbing the Hill Difficulty is the spirit that enables the surgeon to win through every difficulty in the practice of his art.

> This Hill, though high, I covet to ascend;
> The difficulty will not me offend;
> For I perceive the way to Life is here;
> Come, pluck up, Heart, let's neither faint nor fear;
> Better, tho' difficult, th' right way to go
> Than wrong, though easier, where the end is woe.[47]

(*a*) First Stage: The Mastoid Operation: The first stage of the operation, if it has not already been done is to perform very completely the Schwartze-Zaufal operation, during which the descending and horizontal portions of the fallopian canal will be defined. The posterior wall of the osseous meatus is cut away till the fallopian canal is seen;

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44. Robinson, H. P.: The Studio and What to Do in It, 1885, p. 49.
45. Shakespeare: King Henry V, Prologue, l. 22.
46. Greig-Smith: Abdominal Surgery, ed. 5, vol. 1, p. 133.
47. Bunyan: Pilgrim's Progress, Riverside ed., p. 44.

it is also a great advantage to remove much of the floor of the osseous meatus. The latter maneuver exposes, from below and in front, the bend of the facial canal and the lower edge of the horizonal portion. The descending portion of the canal and the part that forms the bend, between the horizontal semicircular canal and the oval window, is surrounded usually by very hard bone, while surrounding the horizontal part of the canal, which forms a bulge on the inner wall of the tympanum, separating the tympanum proprium from the attic, the bone is thinner and more easily removed.

In man there is no doubt that the complete or radical mastoid operation, as already explained, should be done before the operation for removal of the outer wall of the fallopian canal is commenced. As a preliminary to the fallopian canal operation, the first landmark or guide is the definition of the facial nerve at the stylomastoid foramen. When the facial nerve is seen as it issues from the foramen, the topography of the aqueduct and the other stages of the operation present no insuperable difficulties to the surgeon who has practiced the operation on the cadaver or on the living baboon. In the large baboon, however, we have in some instances not completed the mastoid operation, and the mucoperiosteum of the tympanum has been pushed away from behind forward, without opening the tympanic cavity. In other baboon operations, the mucoperiosteum of the tympanum was broken or incised, and the malleus and incus were removed. The stapes was never disturbed. In man, when the complete or radical operation has been done, the stapes, unless diseased, is left undisturbed. After such an operation, completed by the application of a very thin epithelial graft, the hearing power is in large measure conserved. The aim of the surgeon is not only to cure the facial palsy, but also to preserve the hearing.

(*b*) Second Stage: The Operation on the Aqueduct of Fallopius: The second stage of the operation is the removal of the outer wall of the aqueduct of Fallopius. This is a delicate maneuver, and time should not be spared in order to avoid visible, or invisible (concussion), damage to the facial nerve. The instruments for this purpose should be so selected that with them the surgeon might remove the shell from an egg, piece by piece without damage to the vitelline membrane; but, even so, more care is necessary in taking away the outer wall of the canal, because in the egg the vitelline membrane is not attached to the shell covering it, while in the fallopian canal the periosteal lining is closely attached to the bony wall.

We advise that the whole extent of the outer wall of the fallopian canal be taken away up to the region of the geniculate ganglion. The exposure of the geniculate ganglion does no harm; the wall over it being

very thin, it is not difficult to expose. In a few cases, such as those of Bell's palsy in which only the outer wall of the lower half of the descending portion of the aqueduct has to be cut away, the removal of less than the entire outer wall will be sufficient, but we are impressed with the paramount importance of decompression. The great object in view when a nerve graft is employed, is to obtain 4 or 5 mm. of uncovered facial nerve above and below the ends of the nerve graft. The periosteum of the outer wall is attached to the neurilemmal sheath of the facial nerve by delicate filaments of fibrous tissue. The sheath should be freed from the bone at the stylomastoid foramen, for at this point it is firmly attached. The outer wall of the fallopian canal should be taken away entirely before the fibrous lining is slit up. In this way, the nerve is protected from injury during the removal of the outer wall of the canal. The periosteum or fibrous sheath is comparable to the dura mater. We hold that there is no effectual decompression of the nerve till this fibrous sheath is slit up. In cerebral decompression, a large craniectomy is ineffectual until the dura has been widely opened, hence the importance we attach to the deliberate opening of the fibrous sheath of the nerve. By this method of operation, the nerve is not displaced from the fallopian canal, and the minimum of damage is done to its vascular connections. When ligation of a great artery in continuity was a fairly common operation, the great surgeons of the past taught that the aneurysm needle should pass round the vessel between the sheath and the outer coat, for between the sheath and the outer coat was a lymphatic space that made the passage of the aneurysm needle easy and safe.[48] The space between the periosteal lining of the aqueduct and the facial nerve is comparable to the space between the sheath and outer coat of a great artery. All the tissues and cells of the body are bathed in fluid. The space between the fibrous sheath lining the aqueduct and the facial nerve is small as compared with the interval between the spinal cord and the arachnoid and dural membranes, but in both instances, fluid separates the respective fibrous sheaths from the nervous element. This is an example of the insulation of the nervous system so lucidly described by Trotter:[49] "Every element of the nervous system is kept from contact with somatic tissues either by the dura or by the neurilemmal sheath, and in this insulation there is no chink except those very restricted ones, which allow of contact for functional purposes." When a craniectomy has been done and a portion of dura removed, the lost dura is before long replaced by a strong fibrous membrane, thus reestablishing the insula-

48. Anderson and Makins: J. Anat. & Physiol., 1890, vol. 25, Ballance, C., and Edmunds, W.: A Treatise on Ligation of the Great Arteries in Continuity, New York, The Macmillan Company, 1891, p. 478.

49. Trotter: Lancet **1**:1252 (June 21) 1924; Brit. M. J. **2**:103 (July 17) 1926.

tion of the nervous tissue. This is paralleled by what happens when the outer wall and fibrous sheath of the facial nerve in the aqueduct are removed. The function of the lost sheath is soon taken over by a newly formed fibrous tissue.

The insulation has a practical bearing, for when the periosteal sheath is stroked by a probe, no spasm of the face muscles occurs, but when the finest surgical needle is caused to puncture the sheath, nervous impulses immediately set up spasms in the muscles of the face. Again, in the performance of the mastoid operation, the surgeon happily proceeds with his work, as the watcher fails to report any movement of the face muscles; but this is fallacious; he may find at the end of the operation that the corresponding side of the face is paralyzed. The reason is that when the facial nerve is suddenly divided by a sharp instrument, no movement of the muscles may occur, or only so slight a movement as may escape even the observation of a conscientious watcher.

We are not in favor of the type of operation described by R. C. Martin,[38] which aims at shortening the course of the nerve by displacing it from the canal—from the geniculate ganglion to the stylomastoid foramen—so allowing the damaged portion to be excised and a direct suture supplied. This ingenious plan causes the nerve to lie across the inner wall of the tympanum. The vascular connections in the aqueduct are broken, but some may remain, which are connected with the periosteal sheath. The new bed cannot compare with the old. We have thought much on this subject and performed operations of this type on the living and dead. The foregoing view is expressed as a deliberate judgment founded on experience and experiment.

Instruments to Be Used in Removing the Outer Wall of the Fallopian Canal: Every surgeon must form his own judgment on this matter. We do not desire to be dogmatic. The surgeon's mind is prone to endeavor to devise special instruments for the carrying out of a new task. It is, however, generally found that special instruments are a mistake. Simplicity of instruments and experience in the use of common instruments are of the first importance.

(*c*) Third Stage: The Introduction of a Nerve Graft: With the nerve exposed in the whole length of the aqueduct, let the damaged part be cut away, the two sections being made with a small sharp knife, such as a cataract knife, without disturbing the proximal and distal portions of the facial nerve. We recommend that the gap thus made be filled with a nerve graft taken from the external respiratory nerve of Bell.[50]

50. Sir Charles Bell was born in Edinburgh in 1774 and died in 1842. He was an artist, scholar, philosopher, anatomist and surgeon. He was appointed Surgeon to the Middlesex Hospital, London, in 1812. He treated many wounded after the battle of Waterloo. The life and labors of John Hunter (1728-1793) must have been an inspiration to him. He discovered the different functions of

The incision in the axilla at once exposes Bell's nerve under the deep fascia, lying on the serratus magnus muscle. When the required length of Bell's nerve has been cut and placed in the fallopian canal, the divided ends in the axilla should be drawn together by a fine suture. Thus there is no risk of permanent paralysis of the serratus magnus muscle. It is seldom that it is necessary or desirable to attach either end of the nerve graft by suture to the facial nerve, and it is seldom that more than 5 mm. of nerve graft will be required to fill the gap. However, if the operation is attempted in a case of facial palsy following operative injuries or fracture of the middle fossa of the base of the skull, or following gunshot injury of the mastoid, a much longer graft may be required.

We were rather intrigued at first by the idea of curing Bell's palsy by the aid of a graft taken from Bell's nerve in the axilla. Recent experiments, however, appear to prove that any nerve, sensory or motor, will function equally well for the purpose, as long as it is of suitable size to lie in the fallopian canal.

The nerve graft fits accurately in the groove that has taken the place of the fallopian canal, and its ends lie in contact with the cut surface of the facial nerve. Further, it is soon firmly fixed in position by the coagulation of the plasma, which clots as soon as the temperature falls a little. The length of the descending portion of the canal in the adult man is 14 mm., while the horizontal part is from 6 to 7 mm. in length. The diameter of the aqueduct, except in the region of the geniculate ganglion where it is wider, varies from 1 to 1.5 mm.

(*d*) The Dressing and After-Treatment: While performing various anastomosis experimental operations, we were accustomed to surround the anastomosis with pure gold leaf or dentist's gold (22½ carat) or pure gold or platinum foil, 1/2,500 inch in thickness.[51] The foil was sometimes caused to form a tube 2 or 3 mm. in diameter and about 30 mm.

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the anterior and posterior roots of the spinal cord, and that the seventh cranial nerve is motor in function and the fifth cranial nerve sensory. The phrenic nerve he named the great internal respiratory nerve. The nerve to the serratus magnus in the axilla he named the external respiratory nerve. The seventh cranial nerve he named the respiratory nerve of the face, for he said that in great emotional states, e. g., fear or anger, all the respiratory nerves were brought into action, and none more so than the respiratory nerve of the face.

51. The forms of gold or platinum employed were: (*a*) Pure gold leaf, but this is friable and not easy to manipulate. (*b*) Dentist's gold, 22½ carat; this can be unfolded and worked in any direction desired. (*c*) Pure gold foil, $\frac{1}{2500}$ inch in thickness. (*d*) Pure platinum foil, $\frac{1}{2500}$ inch in thickness. The platinum foil is a little stiffer to work with than the gold foil. (*e*) Dutch gold leaf, an alloy of copper and zinc. This should not be used. It is cheap but is rapidly absorbed; meanwhile it may possibly set up an electrolytic action, which would hardly benefit an anastomosis.

long. The idea of surrounding the anastomosis with gold leaf was not so much for protection, as in order to find the anastomosis easily, say, six months after the experiment, when the animal was killed. However gently the operation of exposure of the facial nerve is performed, there is subsequently a good deal of scar tissue formed, and when suturing the nerves, as, say, the great occipital or the cervical sympathetic to the facial, we discovered that by threading a tube of gold or platinum over the proximal nerve before suture, the scar tissue pressure on a small nerve was prevented from interfering with the function of the nerve. Pure gold and pure platinum are entirely unacted on by the living cells and fluids, and so are quite innocuous in the living body.[52]

We have been accustomed to place some gold leaf or platinum foil over the nerve graft, and then to bring a flap of the temporal muscle down so as to fill the mastoid operation cavity.[53] When the gauze has to be changed daily and no muscle flap covers the graft, it is a great advantage to have the graft covered by gold leaf. By this plan no disturbance of the graft can take place.

Ney [54] contributed an admirable anatomic account of methods of opening the facial canal. He describes the slitting up of the sheath of the nerve and the use of pedicled temporal muscle flaps. When the nerve is displaced from the canal, he has devised an ingenious method of placing the temporal fascia on the inside, and the muscle on the outside of the nerve.

What reason have we for placing living muscle over the nerve graft, and again, what happens to the graft? When a nerve graft is placed between the cut ends of a nerve in the middle of a limb, it is immediately bathed in the body fluids that nourish the cells of the body. This is the example that we have sought to follow after placing a nerve graft in the fallopian canal. The conduct and fate of nerve grafts are of much interest. We hope to present a study of this subject at another time and in another paper. But we may say this: There is nothing exceptional to describe. The conduct and fate of a nerve graft is the same as the conduct and fate of a bone graft. The bone graft can be seen, by the fluoroscopic screen, to diminish in size for a time, and then to increase slowly in bulk and fashion itself into the likeness of the bone whose function it is to replace. Certain cells live and others

52. Ballance, C., and Edmunds, W.: A Treatise on Ligation of the Great Arteries in Continuity, New York, The Macmillan Company, 1891, chap. 10, Conduct and Fate of the Ligature.

53. Further experience makes us doubt the value of the temporal muscle graft, at any rate as a routine measure. It should be used only in aseptic cases. The more simple the operation, the more likely is success to be attained.

54. Ney: Facial Paralysis and the Surgical Repair of the Facial Nerve, Laryngoscope **32**:327, 1922.

die. Some absorption takes place. So it is with a nerve graft, if placed in favorable surroundings. The employment of Bell's nerve appears for cosmetic reasons better than taking a nerve graft from, say, the descendens noni nerve, since the use of the latter nerve means an incision in the neck; but any living nerve, motor or sensory, as previously explained, will answer the purpose. We have successfully used the reversed facial, unreversed facial, descendens noni, intercostal and intercostohumeral nerves and the external respiratory nerve of Bell. We have been careful in the majority of the experiments not to reverse the graft, but we do not think that reversing or not reversing the graft is important; in either case, it does its job. In the early stage of the operation, the question arose whether an attempt should be made to lift up the mucoperiosteum of the tympanum in the effort not to open the tympanic air cavity. This plan was adopted by Sourdille,[55] in his operation for the cure of chronic deafness; but we think that in the operation described, it has only a theoretical interest and is not a practical proposition. The essential thing is for the surgeon or some highly skilled person to apply dressings daily.

(*e*) A Case of Facial Palsy:

CASE 1.—A girl, born on July 3, 1930, was admitted to the Manhattan Eye Ear and Throat Hospital, on March 16, 1931, with a prominent inflammatory swelling above and behind the left pinna. A senior house officer operated. At the age of 8 months, the outer boundary of the large antrum is formed by the squama, and in this case it was removed. The further operation accidentally destroyed a considerable length of the facial nerve. After the operation it was noted that complete left facial paralysis was present.

On March 18, 1931, an operation was performed by one of us (Dr. Duel). The outer boundary of the attic and the "bridge" was taken away, thus completing the radical operation. With much difficulty the distal stump of the facial nerve was discovered in the parotid gland just before its division into its two main branches. The proximal stump of the facial nerve was defined on the proximal side of the bend of the canal. A 27 mm. graft of Bell's nerve from the axilla was coaxed into position between the severed ends of the facial nerve. The distal end of the graft was sutured with silk no. 000000 to the divided stump of the facial nerve in the parotid gland. The proximal end of the graft rested against the divided end of the facial nerve in the canal at the bend without suture. Platinum foil covered the graft for two weeks, and was then removed, the graft being well covered by healthy granulation. The wound was dressed daily by Dr. Tickle.

On May 18 (61 days after operation) the gross appearance of facial palsy had disappeared. At rest the face was much changed; it was becoming symmetrical. Faradic response was obtained in the orbicularis oris and the levator labii superioris alaeque nasi.

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55. Sourdille: J. Laryng. & Otol. **45**:601, 1930; The Surgical Treatment of Otosclerosis, J. de laryng. (belge), Oct. 31, 1930.

On May 25 (68 days following operation), on gently tickling the left side of the face with the end of a piece of string during sleep, a distinct movement of the left upper lip and left eyelid was observed.

On June 15 (88 days after operation) the face in repose appeared normal. Even when the patient was crying the mouth was held nearly straight. Corneal exposure was equal on two sides. The faradic current produced a definite response in the muscles of the upper and lower lips and in the orbicularis palpebrarum. Evidently, a rapid recovery from the facial palsy was taking place.

On June 28 (101 days after operation), both eyes were perfectly closed during sleep. On tickling the ala of the nose on the left side and the left upper lip, definite contraction of the levator labii superioris alaeque nasi was seen. On awakening, the palpebral fissures were equal. The corneal reflex was present on the left side, but the left lids did not close as quickly as the right lids. When the patient cried, the mouth was held straight.

4. *When Is It Justifiable to Operate?*—If a galvanic response cannot be obtained in the muscles of the face, it is not justifiable to operate. In long-standing cases, the examination may have to be made under an anesthetic. It is useless to heal the nerve when there are no muscular fibers left for it to energize. As soon as a patient with a paralyzed face is seen, he should be advised to obtain a silver or tortoise-shell hook. This is worn at the angle of the mouth, and is suspended by tape passing around the pinna. Sometimes patients prefer a hook fixed to the teeth by a dentist. The object of this measure is to prevent the drooping of the angle of the mouth due to the lengthening of the muscles. The principle is the same as that in extensor paralysis of the hand or foot. The hand or foot is kept dorsiflexed so as to prevent lengthening of the muscles during the healing of the nerve. The hook should be worn for at least six months after the operation. The after treatment in these cases is almost as important as the operation. Skilled massage must be applied every day and galvanism three times a week. In the case of a child the mother will soon learn how to apply the massage, and many older patients use the galvanic battery daily or every other day, sitting in front of a looking glass.

The operation that we advise does away with associated movements.

Many severe cases of facial palsy make only a partial recovery. Some power of voluntary contraction may return, but along with it comes a tonic contraction of the muscles so that the naso-labial and other folds become deeper than on the sound side which being smoother may now be mistaken for the paralysed side. On voluntary or emotional action, however, this mistake is quickly corrected as the appearance is reversed, the contractions on the sound side being of much greater amplitude. There is no remedy for this state.[56]

When a patient with facial palsy is obviously recovering, it is advisable to stop treatment by massage and electricity, lest the recovery be in a sense overdone.

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56. Pershing, Howell, in Billings-Forchheimer: Therapeusis of Internal Diseases, New York, D. Appleton and Company, 1924.

Another unfortunate event that occurs in some of these cases is a spontaneous spasm or tic movement. When the eyes wink, the upper lip muscles contract. This is sometimes called the winking reflex, but in cases that we have seen, the eyelids and upper lip seem to move together. Under normal conditions a continuous stream of nerve impulses travels along the facial nerve. When the nerve has been injured and partial recovery follows, there remains a hindrance to the passage of nerve impulses. These, it may be imagined, accumulate on the proximal side of the obstruction and then suddenly overflow it; hence the spontaneous spasm. These impulses originate at the level of the corpus striatum. They do not originate in the cortex. They are described under the term paleokinetic in contradistinction to impulses that arise in the cortex which are called neokinetic.[57] In the anastomosis operations and in the intratemporal nerve graft operations on baboons we have noticed in a few cases a slight spontaneous twitch. This tic is more prone to occur following an anastomosis operation than a nerve graft operation. We are inclined to believe that after a nerve graft operation there will be comparative freedom from the defect if early operation is carried out. Contraction and spasm following delayed recovery offer cogent reasons against the common practice of postponing operation. Any one who has seen, as we have, the swelling that surrounds the divided end of the decompressed facial nerve or the swelling that surrounds the region of a nerve graft lying in the fallopian canal during the healing process will not doubt the importance of early operation.

In some of the baboons the expression of the emotions, such as surprise, fear and anger, remind one of the expression of similar emotions in man. In the face of the baboon, the finest lines of emotional expression, such as joy, sorrow, pleasure or disappointment, from our observations are not apparent. The expression of the emotions is the best test as to whether the operation has been followed by the harmonious working of the two sides of the face and of the two hemispheres of the brain. In winking, in yawning and in anger in several of our animals it is clear that the two cerebral hemispheres are working in unison. To investigate fully and prove our belief or otherwise in the several points mentioned would necessitate the keeping of the baboons under observation for several years, and this we are unable to do.

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57. Hunt, J. Ramsay: Brain **40**:58, 1917; Two Distinct Physiological Systems for the Transmission of Motor Impulses in Peripheral Nerves, ibid. **41**:302, 1918; The Static and Kinetic Systems of Motility, Arch. Neurol. & Psychiat. **4**:353 (Oct.) 1920.

5. *The Choice of the Time to Operate.*—The accepted time to operate is *now*. No delay is justifiable. Who does not remember the early days of the operation for appendicitis, when patient after patient was lost because of the delay before operating? Physicians had the habit of saying: "This patient must be protected from the surgeon!" It is hardly necessary to add that their patients died! It must have been more than twenty years after the first appendix operation was done, before the profession realized that the one thing necessary was immediate operation. The same is true of facial palsy. We have seen many patients with facial palsy treated by massage and galvanism for one year, for fifteen months, for two years or for three years, in the hope that recovery of the face would take place without operation. Faint hope! In some cases, slight voluntary movement in two or three muscles was seen, and in some a faradic response was obtained in some of the muscles. In all, however, the deformity of the paralysis of the face persisted, and in all instances the treatment proved valueless and all hopes were falsified. It has been said recently that the difficulty in deciding on operation is that we do not know whether the nerve is crushed, divided or merely involved in inflammation. We do not want to know! The question of operation has little to do with how the paralysis was produced. The reason for the operation is that the patient has facial palsy. In all early mastoid cases, inflammatory or caused by direct injury, an operation should be done immediately; the sooner it is done, the easier is the operation, the less the damage to the nerve and the better the condition of the muscles. In some cases of facial palsy which follow the mastoid operation and in which recovery takes place, say, in three months under appropriate treatment, the reaction of degeneration is never present. Evidently no ciusel or other serious injury has been suffered by the nerve. In more severe cases, the changes in the galvanic responses enable the observer to recognize the moment of the commencement of recovery and the successive stages of improvement that may follow. These changes, however, do not give the observer certain information as to how far recovery will proceed or as to whether the recovery will be only partial or nearly complete. This explains the disappointment of many patients. The all-knowledge prophet is not available, "hopes may be dupes" and not seldom "things are not what they seem." In acute appendicitis no one can tell, however astute and learned he may be, if the patient will recover without an operation. So, in facial palsy, if faradic contractility has long been lost, no one can truly foretell the recovery of the patient. Those who postpone operation in cases of facial palsy with faradic response long lost remind one of the children of Ephraim, spoken of by the Psalmist, who, "being harnessed and carrying bows, turned themselves back in the day of battle."[58]

58. Psalm 78, v. 10 (prayer-book version).

6. *Is Suppuration a Reason for the Postponement of the Operation on the Nerve?*—The answer is *no*. The surgeon prefers an aseptic wound or a healed surface, but in our experience a successful anastomosis may be done in spite of slight suppuration or the weeping from healthy granulations. In any given case, the question of the time for operation depends on the judgment of the surgeon.

(*a*) If the paralysis of the face has been caused by a recent injury or by a recent inflammatory process, which an operation will arrest, it is recommended that one operate at once for the relief of the damage to the nerve.

(*b*) If the paralysis has been present, say, six months in a patient with chronic otorrhea, it may be deemed advisable to postpone the operation on the nerve until the tissue around the fallopian canal has the appearance of healthy granulations, just as epithelial grafting may be postponed for a week or ten days for the same reason. On the other hand, in many cases of chronic otorrhea, the surgeon removes all the disease at the time of the operation and may decide to heal the operative cavity at once by covering it with an epithelial graft. Under such circumstances, it would be folly not to proceed to deal with the damaged nerve at the conclusion of the mastoid operation. The points that we again urge are: The earlier the operation is performed, the easier it is to do, and the more certainty there is of complete recovery of the nerve. John Hunter wisely said: "A fixed principle fixes the mind and a doubtful one leaves it no rest." [59] The fixed principle in paralysis of the facial nerve is immediate operation. "Who" says Carlisle—and we may apply his words to ourselves in the presence of a case of facial palsy—"would go about inculcating doubt, unless he were far astray indeed and much at a loss for employment?" [60]

7. *The Causes of Facial Palsy.*—(*a*) Mastoid Disease and Mastoid Operations: In these cases the sooner operation is done, the sooner the patient will recover from his paralysis.

(*b*) Bell's Palsy: Due to cold; e. g., caused by sleeping in a train with the window open, a draught of cold air playing on the face. These cases appear to be of two types: 1. An infective inflammation of the geniculate ganglion causing a descending neuritis, with herpes of the concha and other signs of involvement of the sensory division of the seventh nerve. J. Ramsay Hunt's paper was the first that we read that dealt with inflammation of the geniculate ganglion.[61] 2. An inflam-

59. Hunter, John: Lancet **1**:491 (Feb. 20) 1904.

60. Carlyle: Frederick the Great, book V, chap. 11.

61. Hunt, J. Ramsay: The Symptom-Complex of Acute Posterior Poliomyelitis of the Geniculate, Glossopharyngeal and Pneumogastric Ganglia, Arch. Int. Med. **5**:631 (June) 1910.

mation of the lower half of the nerve in the descending portion of the fallopian canal. Ney wrote: "The lesion is compressive and located in the lower part of the canal. The chorda tympani escapes involvement usually. If it is involved, the lesion is not so high as to produce paralysis of the stapedius muscle.[54]

In a woman, aged 50, seen recently, who suffered five years before from complete frigidaire palsy of the right facial nerve, there was paralysis of the right occipitofrontalis, so that the brow on the right side was on a lower level than that on the left. The right angle of the mouth could be voluntarily drawn up, but when both sides of the mouth were in action, there were asymmetry and marked deformity. Frequent spasmodic twitching movements of the muscles of the right eyelid and right upper lip occurred during the examination. Pressure over the region of the right stylomastoid foramen elicited tenderness, and the patient said that when she was exposed to draughts, discomfort was always experienced in this region. The recovery, so-called, in this case after five years of medical treatment is a failure as compared with the recovery after an anastomosis operation; if a decompression operation had been promptly performed five years ago on the nerve occupying the lower 1 cm. (three-eighths inch) of the vertical portion of the canal, we think that an almost perfect recovery would have followed.

In cases in the geniculate ganglion, we have seen recovery of the paralyzed face in six or eight weeks. But all patients do not recover.[62] The faradic response should be watched, and if its absence persists, a decompression operation should be done without delay. Some may think that in the cases in the geniculate ganglion, decompression will not be so useful as in other types of facial palsy. But the paralysis of the motor part of the seventh nerve is due to the pressure of the swelling caused by inflammation of the sensory part of the nerve. We urge operation in these cases for the early cure of the facial palsy.

(*c*) Fracture of the Middle Fossa of the Base of the Skull;[63] Gunshot Wounds and Stab Wounds; Tumors: To those attached to a general hospital, fractures at the base of the skull are familiar cases. In many of these, the middle and internal ear and the facial nerve suffer damage. The ear symptoms are often overshadowed by those produced by injury to the brain or by injury to vessels, causing hemorrhage. A fracture of the base in the middle fossa may run alongside the petrous or across the base, implicating the tegmen tympani, or it may pass through the petrous, splitting it longitudinally or transversely. Some fractures pass through the tympanum and the osseous meatus. It is obvious that such injuries may and often do involve the internal ear and facial canal. Should the patient survive, there is always a risk

62. Colledge, L.: Proc. Roy. Soc. Med. **20**:1138 (May) 1927.

63. Passow: Die Verletzungen der Gehörorganes, Wiesbaden, J. F. Bergmann, 1905 (An Account of Injuries of the Ear).

that in the further course of such fractures of the skull, suppuration of the labyrinth and meningitis may occur, and these grave consequences sometimes begin a considerable time after the injury.[64] Other cranial nerves besides the facial are sometimes involved in basal fractures. The fracture may run into the foramen lacerum posterius, and cases in which the vagus has been pressed on by bone or clot were recorded by Rawling[65] and by Hilton.[66] Hilton also clearly described a case in which the hypoglossal was injured in the anterior condyloid foramen.[67]

It seems, at first sight, strange that so dense a bone as the petrous should be so often fractured, but its strength is diminished by the numerous excavations and channels that hollow it out, such as the jugular fossa, the carotid canal, the internal auditory meatus and the facial and other canals. The bony box in which the inner ear lies is of great strength and is seldom fractured. If hearing returns, the deafness was probably due to an effusion of blood that has been absorbed. The facial palsy is permanent. The broken ends of the fallopian canal, it is affirmed, are shifted a little in position, so that one end of the broken canal is not exactly opposite the other end. But this is not the real reason of the continued paralysis. The fundamental explanation is that the inflammatory swelling of the damaged nerve is so compressed by the bony tube within which it lies that regeneration and recovery of function are not possible. The inflammatory swelling is not peculiar to the damaged nerve, but occurs as a result of damage in all tissues. The only treatment is decompression, and in such a case as illustrated, decompression might be attempted with or without the use of a nerve graft. But the main difficulty to be overcome in these cases is localization of the site of fracture and determination of the exact place where the nerve is injured. In many cases, with great damage to the temporal bone, the surgeon must perforce return to his old love and perform an anastomosis.

Makins wrote:[68]

Wounds of the mastoid process in war obtain their chief interest in connection with the paralysis of the 7th nerve. The nerve rarely or never escaped,

64. Weber (Monatschr. f. Ohrenh., 1869, no. 8) reported a case which ended fatally with labyrinthine suppuration twenty-one months after injury. Rovsing (in Chipault: L'État actuel de la chirurgie nerveuse, Paris, J. Rueff, 1902, vol. 2, p. 9) reported a successful operation for infected fracture of the petrous.

65. Rawling: Lancet **1**:1098, 1904.

66. Hilton: Lancet **1**:24 and 147, 1853.

67. Hilton: Lancet **2**:617, 1853.

68. Makins: Surgical Experiences in South Africa, London, H. Frowde, Hodder & Stoughton, 1913, p. 299.

and, so far as my experience went, the facial paralysis was permanent. I think the same prognosis holds good with regard to the deafness resulting from these injuries, and it is difficult to believe, with our experience of the effect of vibration on other nerve centres and organs, that the internal ear could ever escape permanent damage.

Illustrative Cases: CASE 2.—At the age of 15 years, Miss X. fell through the skylight of a conservatory, a distance of over 15 feet (4.5 meters), and sustained a fracture of the base of the skull, with right facial palsy and deafness of the right ear. The deafness cleared up in about one year and a half, but the facial palsy remained complete. Two years and eleven months after the accident, spinal accessory-facial end-to-end anastomosis was performed (in April, 1902).[69]

CASE 3.—Following a bullet wound of the mastoid process in a man, Harvey Cushing [17] performed a spinal accessory-facial end-to-end anastomosis in May, 1902.

CASE 4.—In a man, following a gunshot wound of the temporal bone, Frazier performed an end-to-end anastomosis of the facial and hypoglossal nerves.[30]

CASE 5.—A case came under the observation of one of us (Sir Charles Ballance) many years before this paper was written. The man had received a charge of buckshot on the side of the head in an attack by robbers in South America. The pinna and scalp were riddled by the shot and there was bleeding from the ear. The patient remained unconscious for several hours. When he was seen, six months after the adventure, there was profuse otorrhea, with complete facial palsy. The palsy had been present since the time of the injury. The pinna and adjacent portion of the scalp were much scarred. The mastoid was widely opened, numerous leaden pellets were picked out of the temporal bone, and two were found embedded in the bone forming the medial wall of the tympanum; one of them had divided the horizontal portion of the fallopian canal. The patient made a good recovery, but refused an operation for the cure of the facial palsy. The case was a fine one for a decompression operation, with probably the use of a nerve graft, but in those far-off days, the second-best operation would have been performed, namely, an anastomosis with another nerve.

Stab wounds of the neck may divide the main trunk of the facial outside the stylomastoid foramen. One such case was seen by one of us (Sir Charles Ballance): The ends of the divided nerve were brought together by suture. Free bleeding occurs in these cases. The hemorrhage is arrested and a transfusion may be necessary. Unless the patient is in extremis, the ends of the divided nerve should be sought for and sutured. The earlier the operation for suture of the nerve is done, the easier it is to perform the operation.

CASE 6.—(Tilmann [21]).—A man had had the facial nerve severed at the stylomastoid foramen. The operation of hypoglossal-facial end-to-end anastomosis was done (in February, 1905), five months after the nerve had been severed. It is not clear why an anastomosis operation was chosen or why an end-to-end suture was not attempted.

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69. Ballance (footnote 5, p. 583).

CASE 7.—A man, aged 21, was struck by a glass bottle, which inflicted a deep incised wound in front of the left ear. Immediate peripheral complete paralysis of the left facial nerve occurred. In November, 1902, Dr. W. W. Keen[70] united the distal end of the divided facial nerve to the spinal accessory nerve.

Tumors of the temporal bone and tumors of the parotid region may cause facial palsy.

CASE 8.—Mr. B., aged 44, was admitted to the hospital, with a tumor in the right parotid region, really of the body of the lower jaw. There was complete facial palsy. Nearly the whole of the right lower jaw was removed, and the facial nerve, divided at the stylomastoid foramen, was united end to side to the hypoglossal nerve (in December, 1902).

CASE 9.—B. P., aged 37, was admitted to the hospital with complete right facial palsy. Twenty-one months previously a large parotid tumor had been removed at some other hospital. Since that time, facial palsy had been complete. In January, 1905, an end-to-end anastomosis of the facial and hypoglossal nerves was carried out. A good plan, which we have followed on many occasions, is to dissect out and define the facial nerve, before adopting the steps necessary to remove a simple parotid tumor.

CASE 10.—A woman, aged 32, was admitted to the hospital, under Dr. Hughlings Jackson in 1900, with paralysis of the seventh, eighth, ninth, tenth, eleventh and twelfth nerves. The paralysis of the face had been present for two and a half years. There was a slight foul discharge from the ear, and a polypus was found protruding into the concha. There were no mastoid signs. For four months, severe pain had been suffered. For six weeks there had been hoarseness and difficulty in swallowing. Clearly the case was one of tumor. On operation, only the outer shell of the mastoid was left; the petrous had disappeared. A good deal of hemorrhage occurred during the removal of the tumor, which on microscopic examination was shown to be alveolar sarcoma. The growth was adherent to the dura in the region of the absent posterior and anterior surfaces of the petrous. Five years after the operation, the growth recurred and was again successfully removed. An alveolar sarcoma is apparently only locally malignant. This is an example of a case of facial palsy in which it is not justifiable to attempt a cure by operation.[71]

CASE 11.—One other similar case was seen with Mr. Dolbey in Cairo, three years before this writing. The patient was 55 years of age. He was a prince among the fellaheen and a magnificent specimen of a man. He had a long patriarchal beard, and seemed like Abraham come to life again! The history, the paralysis and the polypus of the meatus exactly reproduced the particulars of case 10. Very little pain had been suffered, and it was found that the patient had slight albuminuria and glycosuria. No operation was done.

Malignant tumors causing facial palsy contraindicate any operation for the relief of the palsy until the time arrives that the surgeon is given the power to control and cure these dread diseases.

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70. Keen: J. Nerv. & Ment. Dis. **30**:368, 1903 (reported before a meeting of the Philadelphia Neurological Society, February, 1902).

71. Ballance (footnote 5, plate 52, fig. 4).

PROTOCOLS OF THE EXPERIMENTS

FIRST SERIES

1. The Anastomosis of the Facial Nerve with the
   (*a*) Hypoglossal Nerve: Experiment 1
   (*b*) Descendens Noni Nerve: Experiment 2
   (*c*) Glossopharyngeal Nerve: Experiments 3 to 8
   (*d*) Lingual and Chorda Tympani Nerves: Experiment 9
   (*e*) Auriculotemporal Nerve: Experiment 10
   (*f*) Superior Laryngeal Nerve: Experiment 11
2. The Substitution of a "Nerve Graft" for a Portion of the Facial Nerve in the Fallopian Canal
   (*a*) Certain Lengths of Facial Nerve "Reversed": Experiments 12 and 13
   (*b*) Intercostal Nerve: Experiment 14
   (*c*) Descendens Noni Nerve: Experiment 15
   (*d*) External Respiratory Nerve of Bell: Experiment 17 (experiments 5 and 17 were performed on the same animal)
3. The Division of the Facial Nerve in the Fallopian Canal: Experiment 16

EXPERIMENT 1.—Medium-sized female baboon, weighing 50 pounds (22.6 Kg.). Operation on Aug. 21, 1930: Hypoglossal-facial end-to-end anastomosis and lingual-hypoglossal end-to-end anastomosis (right side). Suture, catgut no. 0000.[72] Anastomoses surrounded with gold leaf. Discovery of nerves presented no difficulty. Anastomoses made without tension (fig. 12*A*). Removal of facial nerve from fallopian canal was left to be done at some future date.

September 21: Right pouch not used. Right eye did not close.

September 29: Monkey anesthetized (39 days). Faradic Current (strong):[73] On right side, slight but definite response in all muscles. No response with moderate current. Galvanic Current (8 ma., anodal closure): Muscles responded quickly on both sides. Facial Nerve Removed from Fallopian Canal: Mastoid process was opened; also the facial canal. After removal of facial nerve, the stylomastoid foramen and mastoid operation cavity were filled with dentist's gold. Examination of Tongue: Tip pointed to right. Right half of tongue was flatter than left, which was more convex and rotund.

November 2: Marked movement of right side of face in eating.

December 8: Same report as on November 2.

March 3, 1931: Monkey anesthetized (194 days): Same atrophy of right side of tongue as noted on Sept. 29, 1930. No reaction of this side of the tongue to

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72. In these experiments the suture of small nerves was effected: (*a*) By Davis & Geck's black silk no. 000000. The needle had no eye; each suture was affixed to the needle. The needle was straight: from three-fourths to three-eighths inch in length and 0.014 inch in diameter. (*b*) By Davis & Geck's catgut nos. 0000 and 00000. The needles were eyeless, the catgut being affixed to the needle. (*c*) By Allen & Hanbury's fine black silk no. 000000. The needles were straight and curved, and of the thickness of no. 31 standard wire gage. (*d*) By Allen & Hanbury's extra fine synthetic silkworm gut on a very fine ophthalmic eyeless needle.

73. A Wappler portable galvanic and faradic battery was employed for the stimulation of muscles and nerves. For galvanism, from 4 to 6 ma. of current was usually used; for faradism, the regulator bar was usually adjusted to mark 4 or 6.

faradic current. Faradic Current: Response much the same on both sides around mouth and in orbicularis palpebrarum. No response obtained in occipitofrontalis. Galvanic Current (6 ma., anodal closure): Quick small contractions, fairly equal on both sides.

Comment.—The hypoglossal-facial anastomosis caused the complete recovery of the muscles of the paralyzed face. The absence of response in the occipitofrontalis may have been due to some fault in the application of the electrical stimulus. Associated movements of the face with the movements of the tongue, as in swallowing, are a marked sequela of this anastomosis. After the lingual-facial anastomosis the tongue (right side) showed no sign of recovery. We are not, however, without hope that this may yet be observed. In a case seen eight years after this anastomosis was done, the paralyzed side of the tongue was recovering.[74]

April 25: Dr. Beattie's[75] report (247 days). Eyes: In winking, closure of left eye more complete; right eyelids moved sluggishly; winking asynchronous. Ear: No movements visible. Nose: Muscles mobile on right side. Nostrils deviated to right. Mouth: Normal and symmetrical; animal tended to use left pouch more than right. Tongue: Deviated to right when protruded. Associated Movements: In chewing, movements of all facial muscles on right side were seen, such as winking and elevation of ala of nose and angle of mouth.

June 22: Monkey anesthetized (305 days): Tongue: Anterior third of right half of tongue showed no appreciable atrophy. Faradic current produced distinct response in anterior half of right side of tongue, but none in posterior half of right side; normal quick response in left side of tongue. Muscles of Face: Faradic current and galvanic current (6 ma.) produced normal response in all muscles on right side.

July 1 (314 days): Face at rest symmetrical. No spasm or so-called overaction seen. Nose was pulled slightly to the right in feeding. Left pouch used. When animal cleaned teeth with tongue, or in eating, right eyelids closed, and right side of upper lip and right pinna moved.

Comment.—This case presented a fine anastomosis result, with the inevitable associated movements.

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74. Ballance (footnote 5, p. 575).

75. There is a divergence between the reports on the monkeys, showing recovery of normal electrical reactions of the muscles of the face, and the reports of Dr. Beattie, associate professor of anatomy in McGill University, Montreal, who watched the animals moving about the cages and eating. The normal movements of the two sides of the face depend on the two cerebral hemispheres functioning in unison. The stage of a perfect recovery is the result of a reeducation of associated movements. It requires a much longer period of time to perfect than the recovery of normal faradic response in the muscles of the previously paralyzed side of the face. The recovery of synchronous movements of the muscles of the two sides of the face occurs sooner after an intratemporal operation than when the facial is grafted on to another nerve. Indeed, in the latter case, it may not happen at all.

EXPERIMENT 2.—Medium-sized male baboon, weighing 30 pounds (13.6 Kg.). Operation on Aug. 22, 1930: Descendens noni-facial end-to-end anastomosis and hypoglossal-descendens noni side-to-end anastomosis (right side); facial nerve removed from fallopian canal for 12 mm. Suture, black silk no. 000000. Needle, straight, very fine, three-eighths inch (0.9 cm.) in length. Anastomosis surrounded by gold leaf. Facial nerve was exposed. Descendens noni was divided and its proximal end united to trunk of facial nerve. Distal end of descendens noni nerve was anastomosed, end to side, to the lower border of the hypoglossal nerve. This anastomosis was done because previous experience had taught that it was always successful in preventing permanent atrophy of the three muscles supplied by descendens noni. One suture was used in uniting descendens noni to facial nerve, and two sutures for side union of hypoglossal and descendens noni nerves. Suturing accomplished without tension. Mastoid operation was done last; more than one-half inch of nerve removed from fallopian canal.

September 21: Typical signs of palsy present: Right eye did not close; nose was pulled to left, and left food pouch only was used.

October 12: Condition same as on September 21.

October 13: Monkey anesthetized (52 days): Right eye did not close; mild conjunctivitis present; bathed with colloidal silver solution. Faradic Current: On right side, definite slight response in all muscles around mouth. Slight flicker in orbicularis palpebrarum.

November: Dr. F. T. van Beuren present. Marked associated movement of pinna during eating.

December 6: Movement of right pinna marked during swallowing.

Feb. 13, 1931: Monkey anesthetized (175 days): Faradic Current: Good response all around mouth, but not equal to that on left side. No contraction of occipitofrontalis. Galvanic Current (6 ma., anodal closure): Quick response on both sides, but slightly slower and wider contractions on right.

Comment.—It appears that in less than six months the recovery of the paralyzed face made good progress. Especially notable are the associated movements during eating.

April 25: Dr. Beattie's report (246 days). Eyes: Occipitofrontalis moved equally on both sides. Palpebral fissures equal. Winking movements synchronous. Ear: Movements normal. Right ear turned somewhat forward. Nose: Left side moved more than right, but movements of right present. Mouth: Opening normal. Left pouch filled first. Associated Movements: (*a*) closure of right eye complete; (*b*) movement of right ear; (*c*) movement of right nostril; (*d*) movement when animal yawned. Tendency of left side of face to move synchronously with right.

June 6: Monkey anesthetized (288 days). Faradic Current: Muscles around mouth responded well on both sides. Galvanic Current (6 ma., anodal closure): Response equal on the two sides. During recovery from anesthesia (partial anesthesia), spontaneous spasm of muscles of whole right side of face were observed. On stimulation of muscles on right side of upper lip, both upper and lower lip contracted. Corneal reflex less active on right than on left side.

July 1 (313 days): No spasm observed. Face symmetrical. Winking of right lids lagged behind that of left. Associated movements of right pinna and right side of upper lip.

Comment.—This case shows a good result of an anastomosis operation, but with the inevitable associated movements.

EXPERIMENT 3.—Medium-sized female baboon, weighing 25 pounds (11.3 Kg.). Operation on Aug. 12, 1930: Glossopharyngeal-facial end-to-end anastomosis (right side). Two sutures, black silk no. 000000. Anastomosis surrounded by muscle; adjoining muscles sutured around it. Glossopharyngeal and facial nerves defined by dissection in usual manner. Facial nerve was divided at the stylomastoid foramen and turned forward. Glossopharyngeal nerve was severed far forward and turned backward. Anastomosis effected without tension. Fallopian canal was opened, and nerve removed from stylomastoid foramen to the bend. Proximal opening of fallopian canal plugged with dentist's gold, and mastoid operation cavity in part filled with same material; over this a sheet of sternomastoid muscle was fitted. Distance from bend of fallopian canal to anastomosis, 2.5 cm.

September 21: Little deformity during repose.

November 2: Both eyes winked. Animal constantly moving; impossible to observe for associated movements; very little left pull of the nose.

November 16: Monkey anesthetized (96 days). Faradic Current: Slight but definite response in all muscles around mouth. With stronger current good response in all muscles around mouth. Galvanic Current (7 ma., anodal closure): Much wider muscular contractions on right side than on normal side.

December 6: Constant rushing about of monkey prevented reliable observation in regard to associated movements during the eating of an apple.

Feb. 2, 1931: Monkey anesthetized (193 days). Faradic Current: Very good response in all muscles except occipitofrontalis, in which contraction was slight. Response only slightly less quick than that on left side. Galvanic Current (6 ma., anodal closure): Contractions on right side were a little wider than those on left side.

Comment.—In this case of glossopharyngeal-facial anastomosis the paralyzed face was on the way to recovery. It is much more difficult to observe associated movements during eating in a large monkey that has long hair covering the ears and that is confined in a large cage than in a rhesus monkey in a small cage. A sign not observed is not necessarily a sign not present.

April 25: Dr. Beattie's report (257 days). Eyes: Occipitofrontalis contracted well on each side. Right palpebral fissure normal. Winking movements were seen on right side, but were not strong enough to draw eyelids together. Such movements of right eyelids were asynchronous with the normal movements of left side. Nose: Movements of muscles equal on each side of the nose. Mouth: Normal in position; each angle moved upward and laterally to an equal extent. Ear: Movements apparently normal. Activity of animal prevented a more complete series of observations. Associated movements were well marked in swallowing: (*a*) closure of right eye, (*b*) nose movement and (*c*) backward and forward movement of the pinna.

June 6: Monkey anesthetized (299 days): During partial anesthesia, spasm of all muscles of right side of face, especially around the mouth. Such movements occurred when cheek was stroked, showing that reflex arc was intact. Excursions of muscles were not wide and were not unlike the associated movements seen during swallowing. Harmonious action of the two cerebral centers apparently not in working order. Faradic Current and Galvanic Current: Normal responses, equal on both sides.

July 1: No spasm seen. When animal was nervous, upper lids did not move synchronously but both moved actively. Condition apparently was not so good as formerly. Associated Movements: On closure of right eye, movements of right side of upper and lower lips and right pinna occurred.

Comment.—The animal made a good recovery except for the apparently inevitable associated movements.

EXPERIMENT 4.—Medium-sized male baboon, weighing 39 pounds (17.7 Kg.). Operation on Aug. 13, 1930: Glossopharyngeal-facial end-to-end anastomosis (right side). Suture, black silk no. 000000. Anastomosis surrounded by muscle. Glossopharyngeal nerve was first sought. Carotid artery, jugular vein and vagus nerve were followed to the base of the skull, when petrous ganglion of glossopharyngeal came in sight. Glossopharyngeal nerve was stimulated and stylopharyngeus contracted. Nerve was divided far forward and turned backward. Facial nerve severed at stylomastoid foramen and turned forward. Trunk of facial nerve measured 8 mm. Nerves brought together without tension. Mastoid process opened, and facial nerve removed as far as the bend. Gold leaf was used to plug opening of fallopian canal at the bend.

September 21: Little deformity except during eating.

October 12: Used left pouch only (?). Some associated movement. Observation difficult; animal in constant motion in cage.

October 17: Monkey anesthetized (65 days). Faradic Current: Well marked contraction of upper lip. Slight contraction of muscles of lower lip. Very slight response of orbicularis palpebrarum.

November 2: Winking of both eyes almost equal. Right eyelids lagged slightly behind left. Few other signs of palsy. Impossible to observe for associated movement.

March 2, 1931: Monkey anesthetized (201 days). Eyelids winked equally. Faradic Current: Equal response on both sides in all muscles. Galvanic Current (6 ma., anodal closure): Equal response on both sides in all muscles.

Comment.—In this case a glossopharyngeal-facial anastomosis resulted in a good recovery from complete facial palsy. The glossopharyngeal nerve, with the exception of a minute motor branch to the stylopharyngeus, is wholly sensory in function.

April 25: Dr. Beattie's report (255 days). Eyes: Left occipitofrontalis moved more than right, and was asynchronous with right. Palpebral fissures equal. In winking there was complete closure only with left eye. Ear: Not observed. Nose: Moved slightly to right. Mouth: Normal opening. Right pouch mostly used. Associated Movements: Not observed. Recovery of lower part of face.

July 1 (322 days): Both occipitofrontales worked well; perhaps left showed a little stronger movement. Palpebral fissures equal. Winking equal. Mouth normal. Slight spasmodic movement seen above the right angle of mouth. This was frequently repeated but had no relation to winking. Monkey anesthetized. Spasmodic movement above right side of upper lip (levator anguli oris and levator labii superioris alaeque nasi); exaggerated under ether. Faradic Current: On right side, all muscles contracted well. Occipitofrontalis and orbicularis palpebrarum same as on left side. With strong current (electrode over trunk of nerve), all muscles contracted equally with those on left side. Galvanic Current (6 ma., anodal closure): Both sides responded normally. Possibly a little more contraction on right side.

July 2: No spontaneous movement and no associated movements observed. Animal in constant movement, which made observation difficult.

Comment.—Recovery took place without noticeable associated movements.

EXPERIMENT 5.—Large male baboon, weighing 50 pounds (22.7 Kg.). Operation on Aug. 14, 1930: Glossopharyngeal-facial end-to-end anastomosis (right side). Suture, black silk no. 000000. Anastomosis surrounded by muscle. A long, almost horizontal portion of the glossopharyngeal was easily exposed. Petrous ganglion came into view. Nerve was stimulated as it crossed the internal carotid artery; stylopharyngeus muscle contracted. Nerve was divided in front, turned backward and united without tension to divided end of facial nerve. Less disturbance of parts ensues if nerve is passed backward below posterior belly of digastric muscle than if it is inveigled through tissues at upper border of this muscle; generally there is ample length of nerve to make this plan feasible. Mastoid process was large and pneumatic. Canal of facial was opened as far as the bend and the nerve removed. "Groove" caused by opening of fallopian canal was filled with gold leaf. The vertical portion of fallopian canal was 12 mm. in length. Trunk of facial nerve was 15 mm. long, and distance of bend to anastomosis was 3.5 cm. Stimulation of glossopharyngeal caused right side of pharynx to move upward and outward, and the back of right side of tongue was pulled slightly in same direction.

September 21: Little deformity at rest, but obvious when animal was eating.

October 14: Less general appearance of palsy than on September 21.

October 19: Monkey anesthetized (66 days). Dr. H. S. Pritchett present. Faradic Current: On right side, definite but slight response in muscles of upper lip. Very slight response in lower lip and in orbicularis palpebrarum. Galvanic Current (6 ma., anodal closure): On right side, marked wide contractions of muscles around mouth; very different from quick small contractions on normal side.

November 2: Eyelids winked equally. Nose not pulled to left. Movements of monkey made observation for associated movements impossible.

December 6: Almost complete symmetry of face during eating.

March 4, 1931: Monkey anesthetized (202 days): Faradic Current: On right side, all muscles around mouth reacted moderately. No response in orbicularis palpebrarum or in occipitofrontalis. Galvanic Current (6 ma., anodal closure): On right side, wider excursions of muscular contractions around mouth than on left side.

March 8: Monkey anesthetized for a nerve-grafting operation on left facial nerve. Opportunity was taken to stimulate right side of face again. Previous results confirmed.

Comment.—It is not yet obvious why the recovery of the lower part of the right side of the face is not more advanced or why there is no response to stimulation in the orbicularis palpebrarum and occipitofrontalis. Time may solve the problem.

EXPERIMENT 17.—Same baboon as in experiment 5. March 8, 1931: Second operation (left side). Outer wall of fallopian aqueduct removed from stylomastoid foramen to region of geniculate ganglion. Ten millimeters of Bell's nerve (one branch to a serration of the serratus magnus) substituted for 10 mm. of left facial nerve removed from fallopian aqueduct. Graft covered by a sheet of platinum foil, $\frac{1}{2.500}$ inch in thickness; mastoid cavity then filled with a pedicled

graft from temporal muscle. Nerve graft extended from 2 mm. above stylomastoid foramen to just beyond the bend. No sutures used. Nerve graft fitted accurately in "gutter," and its ends impinged on cut surfaces of facial nerve.

April 25: Dr. Beattie's report (right side, 254 days; left side, 48 days). Observations difficult because of movements of animal. Eyes: Occipitofrontales did not seem to move. Palpebral fissures equal. Winking not observed. Ear: No movement seen. Nose: Movements seen on right side. Mouth: Drooped slightly on right side. This part of face moved out of time with left side. Associated Movements: Not seen. Apparently partial recovery of right lower part of face.

June 23: Monkey anesthetized (right side, 313 days; left side, 107 days). Corneal reflex on right side; none on left. Faradic Current: No response of occipitofrontales; response of orbicularis palpebrarum on left side; no response on right. Small contractions around right side of mouth. On left side of mouth, good response, especially of the levator labii superioris.

July 3: Left side of face showed better recovery than right. Animal used both pouches. Protective wink on both sides, but quicker on left than on right.

Comment.—There seemed to be an arrest of recovery of the right side of the face (glossopharyngeal-facial anastomosis), while the recovery of the left side (nerve graft operation done much later) had overtaken that on the right side, and in certain aspects had even passed it.

July 24: Monkey anesthetized (right side, 344 days; left side, 138 days). On right side, frequent twitch of upper and lower eyelids. Slight contraction of angle of mouth and contraction of the levator menti. These movements were associated with similar movements of posterior half of right side of tongue. Spasmodic contractions were not observed before anesthesia. No spasmodic contractions of left side of face and tongue. Faradic Current: All muscles on both sides responded. Response a little better on left side. Galvanic Current (6 ma., anodal closure): On left side, quick small normal contractions. On right side, slower and larger movements.

EXPERIMENT 6.—Large male baboon, weighing 50 pounds. Operation on Aug. 15, 1930: Glossopharyngeal-facial end-to-end anastomosis (right side). Suture, finest black silk no. 000000. Anastomosis not surrounded by muscle, gold or platinum. Trunk of facial nerve (one-half inch in length) defined. Was divided at stylomastoid foramen, and cut end was turned forward. Glossopharyngeal nerve found underneath posterior body of digastric muscle; was divided before it entered the muscle in front. Thus a comparatively long piece of the nerve was turned backward and carried through tissue at upper border of digastric muscle. Suture of the two nerves was effected without tension. Facial nerve in the fallopian canal removed from the stylomastoid foramen to a point just beyond the bend. Facial nerve taken from its canal measured over one-half inch. The "groove" and in part the mastoid operation cavity were filled with dentist's gold.

September 21: Well marked signs of facial paralysis.

October 21: Monkey anesthetized (71 days). Faradic Current: Slight but definite response in muscles around mouth and in orbicularis palpebrarum.

November 2: Definite associated movement of pinna observed.

December 6: Same report but observation difficult in consequence of constant sudden movement and long hair of animal.

Feb. 23, 1931: Monkey anesthetized (192 days). Faradic Current: Contractions of muscles around mouth nearly equal, but those on right side not quite as vigorous as those on left. Response in orbicularis palpebrarum. None in occipitofrontalis. Galvanic Current (6 ma., anodal closure): Quick movements in all muscles around mouth on both sides. Contractions on right side were slower and wider than those on left.

Comment.—In this monkey the glossopharyngeal-facial anastomosis had resulted in partial recovery of the paralyzed face.

April 25: Dr. Beattie's report (253 days). Eyes: Sluggish winking movements of right lids. Winking of right eyelids not complete, but movement of both sides was synchronous. Ear: Right ear moved backward on swallowing. Nose: Deviated to right. Slight flattening of facial contour posterior and lateral to the nostril. Mouth: Lower lip dropped somewhat, but to same extent on both sides. Both pouches used. Lips used to same extent on both sides. No deviation to right or left.

June 23: Monkey anesthetized (312 days). Faradic Current: On right side, small contractions of muscles around mouth as compared with left side. No response obtained in occipitofrontalis and in orbicularis palpebrarum. Galvanic Current (6 ma., anodal closure): Wide wavelike contractions on right side around mouth as compared with left side. No spontaneous contractions.

July 1 (320 days): Winked left eye only. No protective closure of right eye. Both pouches filled. Nose straight. Right lower lip drooped. No spasm seen.

Comment.—Incomplete recovery of the right lower part of the face took place.

EXPERIMENT 7.—Medium-sized female baboon, weighing 40 pounds (18.1 Kg.). Operation on Aug. 16, 1930: Glossopharyngeal-facial end-to-end anastomosis (right side). Suture, black silk no. 000000. Anastomosis not surrounded by muscle. Facial nerve divided at stylomastoid foramen and turned forward. Trunk of facial was more than one-half inch in length. Tendon of digastric muscle divided between sutures. Glossopharyngeal nerve exposed; stimulated with faradic current; mouth was held open and moving pictures were taken of movement of the pharynx caused by contractions of right stylopharyngeus muscle. As posterior belly of diagastric muscle was displaced downward by the severing of its tendon, glossopharyngeal nerve and facial nerve were brought together and sutured at upper and inner border of this muscle. Anastomosis made without tension. Divided tendon of digastric was sutured. Fallopian canal was opened to a point beyond the bend, and long piece of facial nerve removed. "Groove" and much of mastoid operation cavity were filled with dentist's gold.

September 21: Well marked signs of facial palsy.

October 15: Monkey anesthetized (60 days). Faradic Current: On right side all muscles around mouth showed good response. No contraction in orbicularis palpebrarum or occipitofrontalis. Galvanic Current (6 ma., anodal closure): Responses on right side not as perfect as those on left side.

November 2: Associated pinna movements clearly observed.

December 6: Observation difficult. Pinna movements while animal was eating apple.

Feb. 22, 1931: Monkey anesthetized (191 days). Faradic Current: On right side, good quick response in muscles around mouth; almost identical with contractions of those on left. Orbicularis palpebrarum contracted. Occipitofrontalis did not contract. Galvanic Current (6 ma., anodal closure): Quick small normal contractions of muscles around mouth, equal on both sides.

Comment.—Fair recovery had taken place. Galvanic stimulation produced short quick normal contractions, instead of the earlier wide sluggish movements of partial recovery.

April 25: Dr. Beattie's report (252 days). Eyes: Palpebral fissures equal. Winking of right eye synchronous with left, but not as complete. Occipitofrontalis working on both sides, but movement on right was sluggish and smaller in range than on left. Ear: Retraction of right ear on swallowing. Mouth: Lips evenly placed. Mouth opening normal. Left pouch used first, and then right.

Right pouch emptied by pressing with hand. Nose: Nasal muscles used equally. No deviation to either side. Tongue: Normal equal movements.

June 23: Monkey anesthetized (314 days). Faradic Current: Normal and equal contractions on both sides, all muscles responding. Galvanic Current (6 ma., anodal closure): Normal and equal contractions on both sides, all muscles responding. On stroking face, right angle of mouth was pulled upward and outward. These spontaneous contractions occurred also without stroking stimulus.

July 1 (322 days): No spasm noticed on long observation. Associated movements in swallowing, affecting right eyelids, right upper lip and pinna. Right angle of mouth pulled backward. Associated movements not as marked as in hypoglossal anastomosis.

Comment.—This experiment showed a good result of an anastomosis operation with the usual associated movements.

EXPERIMENT 8.—Medium-sized male baboon, weighing 40 pounds. Operation on Aug. 17, 1930: Glossopharyngeal-facial end-to-end anastomosis (right side). Suture, black silk no. 000000. Anastomosis not surrounded by muscle. Facial nerve divided at stylomastoid foramen. Its trunk measured a little more than one-half inch. Glossopharyngeal nerve was found in its forward portion to have divided into two branches, one (motor portion) being very small. These were divided, carried backward and united to cut end of facial nerve. No tension at anastomosis. Mastoid process opened, and facial nerve removed from fallopian canal from stylomastoid foramen to a point a little beyond the bend. Length of opened canal was three-fourths of an inch (1.9 cm.). Distance from the nerve just projecting from unopened canal beyond the bend to anastomosis, 1¼ inches (3.1 cm.). "Groove" and part of mastoid operation cavity filled with dentist's gold.

September 21: Signs of facial palsy well marked.

October 15: Monkey anesthetized (59 days). Faradic Current: Muscles of right side of upper lip showed good response; muscles of right side of lower lip, slight response. Right orbicularis palpebrarum responded with closure of lids. No response in occipitofrontalis. With weaker current, response was slight in upper lip muscles and absent in lower lip muscles.

November 2: Distinct associated pinna movement.

November 12: Monkey anesthetized (87 days). Faradic Current: Slight response of muscles on right side of lip. With strong current, good response in muscles of right side of upper and lower lips.

Feb. 21, 1931: Monkey anesthetized (188 days). Faradic Current: Equal response of muscles on both sides of face. Right occipitofrontalis responded well. Galvanic Current (6 ma., anodal closure): Response nearly equal on the two sides. Contractions on right side perhaps slightly wider.

Comment.—The search for the glossopharyngeal nerve should be conducted along the internal carotid artery. The nerve crosses this vessel and the minimum of dissection is required if the course of the great vessels and vagus nerve is followed. This experiment has resulted in recovery of the baboon from complete right facial palsy. It is to be noted that at the last examination a good response was obtained in the occipitofrontalis muscle.

April 25: Dr. Beattie's report (251 days). Eyes: Occipitofrontales showed equal movements. Right palpebral fissure smaller than left. Winking synchronous and equal in range. Ear: Retraction on swallowing. Nose: Movements of two sides equal. Mouth: Fissure normal and lip movements equal in extent. Tendency to fill left pouch first. Associated Movements: While right side of face and also platysma moved on swallowing.

June 22: Monkey anesthetized (309 days). Faradic and Galvanic Currents: Equal response of all muscles on both sides of face. Under partial anesthesia there was an occasional spasm of muscles of right side of face, which pulled mouth toward that side.

July 1: No spontaneous spasm observed. Face symmetrical. Associated movements of right eyelids and of right angle of mouth, which was pulled backward.

Comment.—This is an excellent result of an anastomosis operation with the usual associated movements.

EXPERIMENT 9.—Large male chacma baboon, weighing 45 pounds (20.4 Kg.). Operation on Aug. 28, 1930: Lingual-facial end-to-end anastomosis (right side). Suture, catgut no. 0000. Anastomosis surrounded with gold leaf. Lingual should be sought far forward at anterior border of hypoglossus muscle, where it is connected by small branches with hypoglossal. It obviously cannot be found except in front of anterior border of pterygoideus internus muscle. The thin sheath of fascia under which it lies makes its discovery difficult unless the fact is borne in mind.[76] Nerve was inveigled backward between inner surface of jaw and outer surface of pterygoideus, about 1 inch above angle of jaw, where pterygoideus separates from inner surface of the jaw. Facial trunk divided at stylomastoid foramen and turned forward. Anastomosis made without tension. As bleeding occurred from lingual artery, and 60 cc. of saline solution had been injected subcutaneously, it was decided to remove facial nerve from fallopian canal at some future date.

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76. Testut (footnote 32, ed. 8, vol. 3, p. 111, fig. 103; p. 118, fig. 111.

September 21: When animal was eating, signs of palsy were evident.

October 11: Monkey anesthetized (44 days). Faradic Current (strong): On right side, definite response in all muscles around mouth. Galvanic Current (6 ma., anodal closure): Equal response on both sides. Facial Nerve Removed from Fallopian Canal: Nerve was identified outside the stylomastoid foramen. It ended in a bulb. Bulb was seized by forceps; with gentle traction, 16 mm. of nerve was drawn out of canal.

November 2: No associated movements in eating.

November 8: Monkey angry. Left side of upper lip raised; no movement of right side of lip.

December 6: Face symmetrical at rest. Palsy was marked while animal was eating.

Feb. 27, 1931: Monkey anesthetized (183 days). Faradic Current: On right side, definite but not wide contraction movements of all muscles around mouth. No response in orbicularis palpebrarum or in occipitofrontalis. Galvanic Current (6 ma., anodal closure): On left side, quick (normal) small contractions. On right side, wide muscular contractions around mouth. Orbicularis palpebrarum showed good response; eyelids closed.

Comment.—This experiment was an anastomosis between the lingual and chorda tympani nerves and the facial nerve. The latter was united to two sensory nerves. The result appears to be a stage in the evolution of recovery of the palsied face. We have noted that while recovery of the muscles is proceeding, the response of the previously paralyzed muscles to the galvanic current is a wide excursion contraction of each muscle.

April 25: Dr. Beattie's report (240 days). Occipitofrontalis moved more on left side. Palpebral fissures equal. Nose: Movements slight, but present on right side. Mouth: Drooping of right side of lower lip. Right pouch not used. Associated Movements: None.

June 5: Monkey anesthetized (281 days). Eyelids under ether closed nearly equally. Faradic Current: On right side, some response in occipitofrontalis and in orbicularis palpebrarum. With stronger current, good contraction of lower lip muscles; all other muscles responded slightly. Angle of mouth was drawn backward. Galvanic Current (6 ma., anodal closure): Much wider and slower excursions of muscles on right side than on left.

July 1 (307 days): Right canine tooth exposed, owing apparently to right side of upper and lower lips being thinner than left side. No drooping of right side of lower lip. No appearance of facial paralysis. Animal used both pouches and chewed on both sides of mouth. Both corneas equally exposed. Protective winking of left lids more rapid than that of right. Left occipitofrontalis showed a little stronger movement than right. No spontaneous spasm of muscles. On yawning widely, mouth was first drawn to right and then a little more toward left.

Comment.—A partial recovery occurred, but the result was much less satisfactory than that following glossopharyngeal-facial anastomosis.

EXPERIMENT 10.—Medium-sized male baboon, weighing 30 pounds (13.6 Kg.). Operation on Aug. 25, 1930: Auriculotemporal-facial end-to-end anastomosis (right side). Suture, catgut no. 0000. Anastomosis surrounded with unfolded dentist's

gold. Auriculotemporal nerve was found above and in front of the meatus on the zygoma. It was followed downward till it passed forward on inner side of neck of condyle of jaw. Nerve was divided as it passed under attrahens aurem muscle, and its proximal segment turned downward to be united to trunk of facial nerve. Anastomosis made without tension. Facial nerve divided at stylomastoid foramen and turned forward and upward. Mastoid operation done, and facial nerve removed from fallopian canal from stylomastoid foramen to a point a little beyond the bend. "Groove," which now replaced fallopian canal, and mastoid operation cavity were filled with dentist's gold.

September 21: Signs of facial palsy well marked.

November 1: Monkey anesthetized (70 days). Dr. F. T. van Beuren present. Faradic Current: On right side, slight but definite response in muscles of upper and lower lips. Doubtful flicking movement in orbicularis palpebrarum. Galvanic Current (6 ma., anodal closure): Response slower and wider on right than on left side.

November 2: Animal used left pouch only. Eyelids winked nearly synchronously, but movement was quicker on the left side. Nose movements equal.

March 1, 1931: Monkey anesthetized (190 days). Eyelids winked almost synchronously, but movements of left eyelids were quicker than those of right. Faradic Current: On right side, quick wide excursions of all muscles around mouth. No noticeable difference from muscular movements on left side. Orbicularis palpebrarum and occipitofrontalis contracted. Galvanic Current (6 ma., anodal closure): Quick small contractions, equal on the two sides.

April 25: Dr. Beattie's report (245 days). Movements of right occipitofrontalis sluggish as compared with those of left. Winking of right eye normal; synchronous with that of left eye. Lips: Position normal at rest. Left pouch filled first. Right pouch was not completely filled and emptied by pressure with hand. Nose: Levator anguli nasi contracted less on right side than on left.

June 2: Monkey anesthetized (283 days). Winking was slightly more rapid on left side. Faradic Current: All muscles responded equally on right and left side of face. Galvanic Current (from 5 to 6 ma., anodal closure): All muscles responded alike on right and left side of face.

July 1 (312 days): Eyelids winked together equally. Nose straight. Right pouch used. Mouth normal. When teeth were shown, left pull of upper lip was slightly stronger than right. No contraction spasm.

Comment.—The auriculotemporal nerve is a sensory nerve. The anastomosis of the facial nerve with this nerve eventuated in recovery from complete facial palsy in this experiment.

EXPERIMENT 11.—Female baboon, weighing 15 pounds (6.8 Kg.). Operation on Aug. 11, 1930: Superior laryngeal-facial end-to-end anastomosis (right side). Suture, catgut no. 0000. Anastomosis surrounded by muscle. Superior laryngeal nerve exposed by following the carotid vessels and vagus nerve upward. The nerve comes off from lower end of ganglion of the trunk of vagus, and passes forward and downward to the thyrohyoid membrane. At this point it was divided, turned backward and united to cut end of the trunk of facial nerve without tension. Trunk of facial nerve was 6 mm. long. Anastomosis was 15 mm. distant from the point in mastoid where facial nerve was divided. Superior laryngeal nerve was electrically stimulated, and the cricothyroid muscle contracted. It will be interesting later to examine atrophy of cricothyroid muscle. Mastoid was opened,

and facial canal was made into a gutter for a length of 7 mm. from the stylo-mastoid foramen. Nerve was removed from mastoid for a distance of 7 mm. Upper opening of the canal was plugged with gold leaf. Mastoid operation cavity was filled with dentist's gold; on surface of the gold a sheet of muscle was adjusted and the deeper tissues sutured over it.

September 15: Monkey anesthetized (35 days). Vocal Cords: Right cord thinner and slacker than left. Abduction movement of right cord was two-thirds that of left. In quiet respiration, right cord lagged behind left, and did not reach middle line in adduction. On touching the glottis, spasm ensued and both cords came up with a snap to the midline.

September 21: Signs of facial palsy well marked.

November 14: Monkey anesthetized (95 days). Vocal Cords: In tranquil respiration, wide abduction of both cords. On touching vocal cords, quick adduction; both cords came together with a snap. Faradic Current: Definite contraction of muscles of upper and lower lips; slight contraction of orbicularis palpebrarum.

Feb. 24, 1931: Monkey anesthetized (190 days). Right and left cords moved equally, in tranquil respiration and in spasm; right vocal cord less tense and straight than left. Faradic Current: Right eyelids did not move; left winked constantly. Definite but slight contraction of all muscles of upper and lower lips. Orbicularis palpebrarum did not contract. Definite slight movement of occipito-frontalis. Galvanic Current (6 ma., anodal closure): On left side, quick response with slight muscular contractions. On right side, response in all muscles, but movement was slower and there were much wider excursions.

Comment.—1. From this and other experiments we found that the cutting of the superior laryngeal nerve causes complete paralysis of the corresponding cord; the cord, too, becomes slack from paralysis of the cricothyroid muscle. The vocal cord slowly recovers the power of abduction and adduction, but there is no recovery of the tension of the cord. 2. The reason why complete paralysis of the cord occurs when the superior laryngeal nerve is severed is perhaps that no afferent impulses reach the brain. 3. The superior laryngeal nerve is in large part sensory in function. It has, however, a minute motor filament running with it to supply the cricothyroid muscle. With this explanation before us it may be noted as remarkable that there is definite evidence of recovery of many of the muscles of the face.

April 25: Dr. Beattie's report (250 days): Right occipitofrontalis did not move. Right palpebral fissure was wider than left. Tears filled lower and medial part of conjunctival sac. No winking of right eyelids was seen. Nostrils drawn toward the left. No movement of muscles controlling right side of nose. Angle of mouth drooped on right side. Right food pouch was not employed.

May 31: Monkey anesthetized (286 days). Vocal Cords: Abduction movement of cords in tranquil inspiration fairly equal, but right cord was slack and lagged slightly behind left when in movement. Faradic Current: On right side, response in all muscles around mouth including levator naris. Slight response in orbicularis palpebrarum. No response in occipitofrontalis.

July 2 (316 days): Monkey used right pouch. No protective wink of right eye. Tears from right eye trickled over face. Right eyelids lagged behind left in winking, with only partial closure of eye. Right corner of mouth drooped. Mouth symmetrical in repose.

Comment.—Although the right side of the face gave the appearance of complete facial palsy, the muscles of the mouth and nose responded to the faradic current. This state has been observed in man following prolonged treatment.

EXPERIMENT 12.[77]—Large male hamadryad baboon, weighing 40 pounds. Operation (right side) on Aug. 27, 1930: After opening of mastoid process, facial canal was defined from stylomastoid foramen to a little beyond the bend. Outer wall of canal removed, turning the canal into a "gutter" from a little above the bend to about 2 mm. from stylomastoid foramen (fig. 7*C*). Facial nerve in fallopian gutter divided in two places, 12 mm. apart. Isolated portion of nerve "reversed" in position, so that the original proximal end was distal and the distal end proximal. Facial nerve proximal to graft projected about 2 mm. beyond opening in canal, so that pressure would not affect the inflammatory swelling that would arise at site of upper anastomosis. Distal end of graft was sutured (black silk no. 000000) to trunk of facial nerve. A three-eighths inch, special straight needle, 0.014 inch in diameter, was used. Nerve transplant and anastomosis were covered with pure gold leaf.

Comment.—The nerve looks small and delicate in the fallopian canal as compared with the main trunk of the nerve outside the stylomastoid foramen. This is because it loses its sheath as it enters the bony canal, just as an artery does in the same circumstances. In either case the periosteum and bone are the protecting elements which function for the sheath.

September 21: Facial palsy well marked, especially in eating.

November 2: Face appeared normal in repose. Eyes winked together, but movements of left lids were quicker.

November 15: Monkey anesthetized (80 days). Faradic Current: No response in muscles on right side of face. With strong current definite response occurred in all muscles around mouth and in orbicularis palpebrarum.

December 6: Slight deformity of mouth in eating.

March 1, 1931: Monkey anesthetized (185 days). Faradic Current: Same response on both sides. Galvanic Current (6 ma., anodal closure): Same response on both sides.

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77. In the nerve graft experiments (12, 13, 14, 15 and 17) the diagrams of operations represent accurately what was done in each case. At first the decompression was only of the nerve graft and of a small portion of the facial nerve above and below the graft. These operations were the first experiments of the kind performed. Later (see experiments 18, 19, 20, 21 and 22) we formed the opinion that the outer wall of the aqueduct should be removed throughout its length, beginning with the opening of the stylomastoid foramen. The mastoid process of the baboon is almost horizontal. In this it differs from the mastoid process of man. Each diagram is not a diagram of an operation on a baboon, but it is a diagram of a similar operation on man. The nerve graft occupies the site it occupied in the aqueduct of Fallopius in the baboon. It was thought that to represent each operation as being performed on man would be of practical value to the reader.

April 25: Dr. Beattie's report (240 days). Eyes: Equal movements of eyelids. Palpebral fissures equal. In winking, there were equal synchronous movements. Right lid dropped when animal yawned. Nose: Tilted a little upward on right side. Movements normal. Mouth: Both sides appeared normal; no drooping. Both pouches used. Ear: Movements normal in amount and character. Apparently a complete recovery.

June 6: Monkey anesthetized (282 days). Faradic and Galvanic Current: Responses of all muscles equal on both sides. Some reflex spontaneous movements of muscles on right side of face around mouth under partial anesthesia. These movements occurred when eyelids were touched.

Comment.—This monkey exhibited recovery of the right side of the face from complete facial palsy.

EXPERIMENT 13.—Large male mandrill baboon, weighing 40 pounds. Operation on right side on Aug. 27, 1930: Facial nerve in fallopian canal divided in two places, 5 mm. apart. Portion of nerve between sections was removed from canal, "reversed" and replaced in canal. Graft occupied upper portion of canal just below the bend. It rested accurately in interval between severed ends of facial nerve. No sutures employed. Mastoid process opened, and outer wall of fallopian canal removed to a point about 1 mm. beyond the bend. Thus canal for some distance was converted into a gutter. Graft was covered with a sheet of dentist's gold. Graft shortened considerably on removal from canal. All living structures retract after division; even the spinal cord does so when divided.

September 21: Marked signs of facial palsy.

October 12: Deformity of face much less during eating. In repose, little palsy noticeable.

October 18: Monkey anesthetized (52 days). Dr. Pritchett, Dr. Keppel and Mr. Franks present. Faradic Current: Definite response to stimulation in muscles of upper lip. Little response in lower lip. Slight contraction of right orbicularis palpebrarum. Galvanic Current (6 ma., anodal closure): Quick contraction of muscles of both sides; on right side the contractions had a wider excursion movement.

November 2: Nose was no longer pulled to left side. Great improvement visible in paresis of right side of face.

March 7, 1931: Monkey anesthetized (192 days). Eyes winked equally before and during anesthesia. Faradic Current: Excellent response in all muscles of right side of face, including occipitofrontalis. Angle of mouth was elevated a little less on right side than on left. Orbicularis palpebrarum of each eye responded equally to stimulation. Galvanic Current (6 ma., anodal closure): Contractions of all muscles on both sides of face quick and equal, except that those on right side showed a wider excursion movement than those on left. Eyelids responded equally with quick small movements.

Comment.—This monkey exhibited nearly perfect recovery of the right side of the face from complete facial palsy.

April 25: Dr. Beattie's report (241 days). Eyes: Movements of occipitofrontalis slight but observed; palpebral fissures equal. Animal winked with right eye, but only at times synchronously with left. Ear: Not observed. Nose: Muscles functioning on right side. Mouth: Muscles moved on right side, but when an exaggerated movement (e. g., showing teeth) was carried out, face was pulled to left. Smaller movements were performed equally. Right angle of mouth drooped a little. Almost complete recovery.

June 28: Monkey anesthetized (305 days). Dr. Ramsay Hunt present. Corneal reflex and winking equal. Faradic and Galvanic Current (6 ma., anodal closure): Equal response of all muscles on both sides. On placing an electrode over stylomastoid foramen region (strong current), contraction of muscles of face occurred. Spontaneous spasm of whole right side of face under anesthetic constantly recurred; as in the infant in case 1 during sleep, but in the infant it was a reflex act following tickling of the face.

July 2: Complete symmetry in repose. When animal made a grimace, left side of upper lip was raised more than right, but this asymmetry was improved. With smaller movements of upper lips, no asymmetry. Slight spontaneous twitch in the right side of upper lip, associated with winking.

EXPERIMENT 14.—Large hamadryad baboon, weighing 45 pounds. Operation (right side) on Sept. 17, 1930: Facial nerve in fallopian canal was divided in two places, 6 mm. apart. Isolated portion of nerve removed. Interval thus caused in length of facial nerve was filled with 6 mm. of sixth intercostal nerve. No sutures were used, as ends of graft in "groove" were closely approximated to severed ends of facial nerve. Care was taken not to "reverse" graft. Facial nerve divided about 2 mm. above foramen. It did not show any appreciable retraction; neither did cut end of proximal part of facial nerve. Facial sheath is firmly attached to bone around stylomastoid foramen; delicate fibrous filaments connect nerve with periosteum through whole length of fallopian canal. Nearly 3 mm. of proximal segment of facial nerve lay exposed in groove above stylomastoid foramen before it touched lower end of intercostal graft.

September 21: Signs of complete facial palsy marked.

November 2: Left food pouch only used. Nose not pulled to left. Winking of eyelids unequal.

November 15: Monkey anesthetized (59 days). Faradic Current: Definite movement of all muscles around right side of mouth except levator nasi and near midline of upper lip. Galvanic Current (8 ma., anodal closure): Wide contraction movements of muscles on right side of mouth as compared with those on left side.

Feb. 28, 1931: Monkey anesthetized (164 days). Faradic Current: Good response in all muscles around right side of mouth, but not equal to that on left side. No response in orbicularis palpebrarum or occipitofrontalis. Galvanic Current (6 ma., anodal closure): On right side, quick movement in all muscles around mouth. Contraction excursions somewhat wider than on left side. Right eyelids closed.

Comment.—This experiment has not lasted as long as the other graft experiments. It must be noted that neither the orbicularis palpebrarum nor the occipitofrontalis responded to the faradic current.

April 25: Dr. Beattie's report (220 days). Right eyelids stationary; no winking. Palpebral fissures equal. Right occipitofrontalis did not move. Nose moved normally. Mouth showed slight drooping of angle on right side. Both pouches used.

June 3: Monkey anesthetized (259 days). Faradic and Galvanic Current (6 ma., anodal closure): Normal contractions on both sides. Good response in orbicularis palpebrarum and occipitofrontalis on right side. Animal winked synchronously on both sides.

July 2: Pulled mouth a little to right.

Comment.—A nearly perfect recovery resulted.

EXPERIMENT 15.—Large dog-faced baboon, weighing 40 pounds. Operation (right side) on Sept. 16, 1930: Facial nerve divided in two places 7 mm. apart in fallopian canal. Piece of nerve between the sections was removed. Interval thus caused in length of facial nerve was filled with 7 mm. of descendens noni nerve. Mastoid process was opened, and the outer wall of the facial canal removed as far as the bend. Graft was placed in canal bed, only one stitch being used (black silk no. 000000) at its distal end, between graft and main trunk of facial nerve at stylo-mastoid foramen. In fitting fragment of descendens noni into canal, care was taken not to "reverse" it. A piece of Dutch gold leaf was spread over the graft, and over this was inveigled a mass of patysma muscle sufficient to fill mastoid operation cavity. Four millimeters of facial nerve, proximal to graft, was exposed from the bend downward. This is important as distal end of proximal segment of a divided nerve forms a bulb. Development of bulb in a rigid canal would cause pressure on nerve fibers and probably lead to failure of experiment (fig. 7 *F*).

Comment.—The use of the Dutch gold leaf was a mistake. This substance is an alloy of copper and zinc, and tarnishes rapidly. We do not know whether it is important not to "reverse" a graft, but we do not think so. The study of this question and our conclusions will be reported at a future date.

November 2: Monkey used left food pouch only. Nose still pulled slightly to the left. Right eyelids winked, but not as quickly as those on left side. Paralysis of right side of face only slightly noticeable.

November 16: Monkey anesthetized (47 days). No winking of right eyelids under anesthesia. Faradic Current: Slight response at angle of right side of mouth. With a moderate current there was a definite contraction of muscles all around right side of mouth. With a strong current, there was a definite contraction of orbicularis palpebrarum. Galvanic Current (8 ma., anodal closure): Marked wide contraction excursion of muscles on right side around mouth as compared with left side. Contractions on left (normal) side were quick, with small excursion movements.

December 6: Monkey showed no signs of deformity in face while it ate a hard substance, e. g., an apple.

Feb. 23, 1931: Monkey anesthetized (160 days). Eyelids of both eyes winked nearly synchronously. Faradic Current: Response to stimulation equal in muscles of right and left sides of face, including occipitofrontalis. Galvanic Current (6 ma., anodal closure): Equal response on the two sides.

Comment.—This monkey exhibited recovery of the right side of the face from complete facial palsy.

April 25: Dr. Beattie's report (233 days). Eyes: Occipitofrontalis showed equal movements as far as one could see; left was perhaps more mobile. Mouth drooped a little on right side; monkey used both pouches. Nose: Tended to flick to left.

June 7: Monkey anesthetized (266 days). Dr. Learmouth's report. Eyes: Winked synchronously and completely. Corneal reflex a little less active on right side. Faradic Current: Good response in all muscles supplied by facial nerve. Movement of frontalis a shade less on right than on left. Galvanic Current (6 ma., anodal closure): Approximately equal responses on both sides.

July 2: Winked evenly. Symmetry of upper lips while eating.

Comment.—Practically a complete recovery took place.

EXPERIMENT 16.—Large baboon, weighing 45 pounds. Operation (right side) on Sept. 19, 1930: Facial nerve divided 6 mm. above stylomastoid foramen. Outer wall of canal removed about 9 mm. upward from stylomastoid foramen (fig. 7 *G*). This decompression would prevent any pressure being exerted on oval swelling that occurs at site of union of severed ends of a nerve. Facial nerve covered with dentist's gold; over this the mastoid cavity was filled by a pedicled graft fashioned from temporal muscle. Distance from stylomastoid foramen to bend was 13 mm.

September 21: Signs of complete facial palsy well marked.

November 2: Right eye lagged behind left in winking. No other evident signs of palsy.

December 6: Monkey anesthetized (78 days). Faradic Current: Slight response on right side in levator labii superioris. Galvanic Current (7 ma., anodal closure): Much wider contraction of muscles on right side than of those on left.

March 1, 1931: Monkey anesthetized (152 days). Right eye winked, but not as quickly as left, under anesthesia. Faradic Current: Good contraction of all muscles. Eyelids closed and occipitofrontalis contracted. Scarcely any difference in contractions of muscles on both sides around mouth. Galvanic Current (6 ma., anodal closure): Short quick movements. No difference on the two sides.

April 25: Dr. Beattie's report (207 days). Occipitofrontalis showed equal extent of movement on right and left sides. Palpebral fissures equal. Nose: Tendency to move to left. Mouth: Left angle raised perhaps a little more than right. Recovery apparently almost complete.

June 5: Monkey anesthetized (248 days). Faradic and Galvanic Current: Responses of all muscles equal on both sides. Slight spasmodic spontaneous contractions of muscles of upper lip noticed while animal was under ether.

July 1 (274 days): Eyelids winked synchronously. Symmetry of face in repose. Examination difficult because of movements of animal. Protective wink equal on the two sides. Slight twitch of right side of upper lip and of orbicularis oris.

Comment.—The experiment is a definite scientific proof of the value of decompression of the facial nerve. If the injury had occurred to a patient and decompression had not been performed, it can hardly be doubted that there would have been permanent paralysis of the face.

SECOND SERIES

1. The Substitution of a Nerve Graft for a Portion of the Facial Nerve in the Fallopian Canal

Experiment 18: Baboon; 16 mm. graft of Bell's nerve
Experiment 19: Baboon; 9 mm. graft of Bell's nerve
Experiment 20: Baboon; 6 mm. graft of Bell's nerve
Experiment 21: Baboon; two 7 mm. grafts of Bell's nerve
Experiment 22: Baboon; 4 mm. graft of Bell's nerve
Experiment 23: Cat; 2 mm. graft of Bell's nerve
Experiment 24: Cat; 4 mm. graft of Bell's nerve
Experiment 25: Cat; 2 mm. graft of Bell's nerve

Experiment 26: Cat; 2 mm. graft of Bell's nerve
Experiment 27: Cat; 2 mm. graft of Bell's nerve
Experiment 28: Cat; 1 mm. graft of Bell's nerve
Experiment 29: Rhesus monkey; 7 mm. graft of Bell's nerve and two 7 mm. grafts of the intercostohumeral nerve
Experiment 30: Rhesus monkey; 7 mm. graft of Bell's nerve

2. The Anastomosis of the Facial Nerve with the
Great Auricular Nerve: Experiment 31 (rhesus monkey)
Transverse Cervical Nerve: Experiment 32 (rhesus monkey)
Great Occipital Nerve: Experiment 33 (rhesus monkey)
Auriculotemporal Nerve: Experiment 34 (rhesus monkey)

3. Experiments Illustrating the Return of Function in a Nerve, a Portion of Which Had Been Excised and Replaced by a similar Length of Another Nerve

Experiment 35: Rhesus monkey; 10 mm. of trunk of facial replaced by 10 mm. of Bell's nerve
Experiment 36: Rhesus monkey; 10 mm. of Bell's nerve replaced by 10 mm. of descendens noni nerve; 10 mm. of descendens noni nerve replaced by 10 mm. of Bell's nerve
Experiment 37: Rhesus monkey; 13 mm. of Bell's nerve replaced by 13 mm. of the internal cutaneous nerve; 13 mm. of the internal cutaneous nerve replaced by 13 mm. of Bell's nerve
Experiment 38: Rhesus monkey; 6 mm. of Bell's nerve excised, reversed and sutured into position
Experiment 39: Rhesus monkey; 5 mm. of the trunk of the facial nerve replaced by 5 mm. of Bell's nerve

EXPERIMENT 18.—Large male hamadryad baboon, weighing 42 pounds (19 Kg.). Operation (right side) on April 4, 1931: Complete radical mastoid operation performed. Facial nerve exposed from geniculate ganglion to a point in parotid where trunk of the nerve divides into its two main branches. A graft of Bell's nerve, 16 mm. long, was implanted in gap caused by removal of facial nerve from the bend to a point 5 mm. outside stylomastoid foramen. Graft was sutured to stump of facial nerve outside stylomastoid foramen with silk no. 000000. Proximal end of graft was coaxed into position without suture. Periosteal sheath had previously been slit up; when graft was in position, edges of sheath were folded over it. Gold leaf was then placed over graft, and a pedicled temporal muscle flap used to fill mastoid cavity. Rubber tube placed in meatus.

April 25: Dr. Beattie's report. Complete facial palsy.

June 27 (84 days): Little sign of palsy in repose. Left eyelids winked quicker than right. Nose pulled to left in eating. Right canine a little exposed owing to drooping of lower lip.

June 28 (85 days): Dr. J. Ramsay Hunt present. No spontaneous contractions of right side of face seen. Faradic Current: Slight response in muscles of right side of upper and lower lips. Galvanic Current (6 ma., anodal closure): Wave-like wide excursion of muscles on right side; normal small contractions on left. When electrode was placed over region of right stylomastoid foramen, there was no response in facial muscles.

July 2: Long observation of monkey in cage and when eating. No sign of facial paralysis.

EXPERIMENT 19.—Large male hamadryad baboon, weighing 44 pounds (19.9 Kg.). Operation (right side) on April 4, 1931: Facial nerve exposed at stylomastoid foramen, and aqueduct opened from this point to 5 mm. beyond the bend. Ossicles not displaced. Radical mastoid operation was not done. Sheath was slit from 1 mm. beyond stylomastoid foramen upward to region of geniculate ganglion. A 9 mm. nerve graft of Bell's nerve was used which corresponded to length of facial nerve excised. Edges of split fibrous sheath were folded over graft, and gold leaf was applied along whole length of graft and of exposed ends of facial nerve. Graft fitted in place without suture. Pedicled temporal muscle graft filled cavity, and covered nerve operation area. Divided ends of Bell's nerve in the axilla were sutured with silk no. 000000.

April 25: Dr. Beattie's report. Complete right facial palsy.

June 27: Monkey anesthetized (84 days). Winked synchronously; left lids closed completely but right lids not quite completely. Corneal reflex on both sides. Faradic Current: Response in orbicularis palpebrarum and in occipitofrontalis. All muscles around mouth gave good response. Galvanic Current (6 ma., anodai closure): On right side wavelike contractions noted in muscles around mouth.

July 1: Observation of monkey in cage and when eating. Face looked normal. No trace of palsy visible. Right canine tooth not exposed. When monkey yawned widely, mouth was slightly drawn to left.

EXPERIMENT 20.—Male hamadryad baboon, weighing 33 pounds (14.9 Kg.). Operation (right side) on April 5, 1931: Modified mastoid operation; ossicles not removed. Facial nerve exposed at stylomastoid foramen to 3 mm. beyond the bend. Sheath was opened from 2 mm. beyond stylomastoid foramen to 3 mm. beyong bend. Six millimeters of Bell's nerve implanted in "groove" in place of a similar length of facial nerve removed. Graft extended from middle of bend downward. No sutures used. Edges of sheath were folded over nerve and graft. Gold leaf was then placed in position from 3 mm. beyond bend to beyond stylomastoid foramen. Over all was inveigled a pedicled temporal muscle graft. Ends of Bell's nerve in axilla were brought together with silk no. 000000. Great auricular nerve cut accidentally, and sutured with silk no. 000000.

April 25: Dr. Beattie's report (20 days). Complete right facial palsy.

July 1 (87 days): Slight drooping of right side of lower lip. No other sign of palsy. No spontaneous spasm. Symmetry on yawning except that right side of upper lip was drawn up more than left. Palpebral fissures equal. Right cornea exposed less than left. Winking not observed. Monkey anesthetized. Faradic Current: Good response all around right side of mouth. No spontaneous contractions. No response when electrode was placed over stylomastoid foramen. Galvanic Current (6 ma., anodal closure): Less response than when negative closure was used; then wider and quicker contractions occurred.

EXPERIMENT 21.—Male hamadryad baboon, weighing 30 pounds (13.6 Kg.). Operation (right side) on April 6, 1931: Pneumatic mastoid opened. Malleus and incus removed. Facial nerve defined entering foramen. Aqueduct was opened from stylomastoid foramen to well beyond the bend. Two 7 mm. lengths of Bell's nerve replaced a similar length of facial nerve removed. Grafts extended from middle of the bend downward. Dentist's gold leaf was laid over grafts and exposed ends of facial nerve. No muscle graft made. Divided end of Bell's nerve could not be easily brought together, so each end was sutured at different places to intercostohumeral nerve.

April 25: Dr. Beattie's report. Complete right facial palsy.

June 27 (82 days): Before anesthesia, face was symmetrical at rest. All gross deformity gone. In eating, nose was pulled a little to left. Slight sagging of right side of lower lip. Monkey ate with no deformity of mouth. Winked evenly. Monkey anesthetized. Winked evenly, but left eyelids seemed to move quicker than right. Faradic Current: On right side, good response around mouth, but better in lower than in upper lip. Galvanic Current (6 ma., anodal closure): Wide contraction of muscles around mouth, most marked in those of lower lip.

July 1: Winked synchronously. Mouth symmetrical during eating. No appearance of facial paralysis.

EXPERIMENT 22.—Large male hamadryad baboon, weighing 35 pounds (15.7 Kg.). Operation (right side) on April 9, 1931: Facial aqueduct opened within the temporal bone. Ten millimeters of Bell's nerve replaced a similar length of facial nerve excised. Graft extended down upper part of vertical portion of aqueduct from the bend. No suture used. Periosteal sheath, which had been slit up, was folded over graft, and a thin strip of gold foil, 1/2,500 inch in thickness, was placed over area of nerve operation. Ends of Bell's nerve in axilla were sutured with silk no. 000000.

April 25: Dr. Beattie's report (16 days). Complete right facial palsy.

July 1 (83 days): Before anesthesia, there was no appearance of gross palsy. Eyes equally exposed. Right side of lower lip sagged slightly. In yawning (mouth opened wide), right side of upper lip slightly more elevated than left. Winking rapid and synchronous, but left eyelids showed a more complete movement than right. Monkey anesthetized. No spasm or overaction observed. Faradic Current: Distinct response in muscles around mouth; less in muscles of lower than in those of upper lip. Galvanic Current (6 ma., anodal closure): Quick response, almost the same on both sides of mouth.

EXPERIMENT 23.—Cat. Operation (right side) on April 25, 1931: Two millimeters of facial nerve in fallopian aquedect replaced by 2 mm. of Bell's nerve.

July 10 (76 days): No appearance of palsy. Palpebral fissures equal. Protective wink on right side. Right eyelids closed. On tickling right side of nose, right side of upper lip was raised. Right pinna moved less than left on tickling. After lapping milk, upper and lower lips moved equally on the two sides as cat cleaned them with tongue. Cat anesthetized. Faradic Current: Stimulation of outer end of transverse fissure (left cortex). Elevation of right side of upper lip and right nostril and closure of right eyelids. Stimulation of right facial nerve in posterior fossa. Well marked contraction of all muscles on right side of face.

EXPERIMENT 24.—Cat. Operation (right side) on April 16, 1931: Four millimeters of facial nerve in fallopian aqueduct replaced by 4 mm. of Bell's nerve.

July 10 (75 days): No appearance of palsy. Palpebral fissures equal. Protective winking. On tickling right pinna, movement not so great as on tickling left pinna. On cleaning lips with tongue after lapping milk, movement of upper and lower lips equal on both sides. Cat anesthetized. Faradic Current: On stimulation of left cortex at outer end of transverse fissure, contraction of right side of upper lip. Closure of right eyelids and movement of pinna. On stimulation of right facial nerve in posterior fossa, contraction of all muscles of right side of face. Lower lip muscles contracted least.

EXPERIMENT 25.—Cat. Operation (right side) on May 3, 1931: Two millimeters of facial nerve in aqueduct of Fallopius replaced by 2 mm. of Bell's nerve.

July 13 (71 days): No appearance of facial palsy. Good protective wink (right eye) with elevation of upper lip and movement of whiskers. Cat anesthetized. Faradic Current: Left cortex stimulation unsatisfactory (battery failing). Pinna moved well, but there was only slight contraction of right orbicularis oris and palpebrarum. Stimulation of right facial nerve at internal auditory meatus produced moderate contraction of all muscles of right side of face.

EXPERIMENT 26.—Cat. Operation (right side) on May 9, 1931: Two millimeters of facial nerve in the aqueduct of Fallopius replaced by 2 mm. of Bell's nerve.

July 11 (63 days): Face appeared normal. On tickling ears, movement of right ear was less than movement of left. Cat anesthetized. Faradic Current: Good response in right side of upper lip and in orbicularis palpebrarum. Less response in lower lip and in occipitofrontalis. Electrode placed over region of trunk of facial nerve. Good contraction of right side of upper lip, orbicularis palpebrarum and right pinna. Stimulation of right facial nerve at internal auditory meatus. Wide pinna movement, but only partial contraction of muscles of upper lip, lower lip and orbicularis palpebrarum.

EXPERIMENT 27.—Cat. Operation (right side) on May 10, 1931: Two millimeters of facial nerve in aqueduct replaced by 2 mm. of Bell's nerve.

July 12 (63 days): No obvious sign of facial palsy. Cat anesthetized. Stimulation of right facial nerve in posterior fossa. All muscles on right side of face contracted. Stimulation of exposed facial trunk just outside stylomastoid foramen; same result.

EXPERIMENT 28.—Cat. Operation (right side) on May 10, 1931: One millimeter of facial nerve in aqueduct of Fallopius replaced by 1 mm. of Bell's nerve.

July 15 (66 days): No sign of facial palsy. Right winking reflex normal. On tickling pinna, reflex response was greater on left side. Cat anesthetized. Faradic Current: On placing electrode over region of right facial nerve (the skin being intact) all muscles on right side of face, including occipitofrontalis, contracted.

Comment.—This cat is being reserved for a few months for further observation.

EXPERIMENT 29.—Rhesus monkey, weighing 4½ pounds (2 Kg.). Operation (right side) on April 1, 1931: Intratemporal nerve graft. Mastoid process opened. Outer wall of aqueduct of fallopius was taken away from region of stylomastoid foramen to middle of bend. Stylomastoid foramen was not interfered with. A 7 mm. graft of Bell's nerve and two similar lengths of intercostohumeral nerve were successively laid in groove lying between cut ends of facial nerve. No sutures were used. Grafts were covered with dentist's gold.

June 3: Monkey anesthetized (63 days). Faradic Current: Good response around mouth. Less in lower than in upper lip muscles. Also response in orbicularis palpebrarum, but none in occipitofrontalis.

July 7 (97 days): Right eyelids did not close during sleep. Right corneal reflex present. When monkey was awake, protective wink was complete. Mouth and nose normal. Protective wink associated with a spasm movement of muscles of lower lip. Monkey anesthetized. Faradic Current: Fine response in upper lip; less response in lower lip. Also response in levator nasi and orbicularis palpebrarum (closure of eyelids). Galvanic Current (6 ma., anodal closure): Both

sides gave equal response. On waking from anesthesia, no spasmodic movements were noticed except when a wink occurred synchronously with movement of lower lip.

July 8: No spasmodic movements seen in long period of observation.

EXPERIMENT 30.—Rhesus monkey, weighing 5 pounds (2.2 Kg.). Operation (right side) on March 30, 1931: Intratemporal nerve grafts. Mastoid process was opened. Outer wall of aqueduct of Fallopius was removed from stylomastoid foramen to a point well beyond the bend. Seven millimeters of Bell's nerve was coaxed into place previously occupied by a similar length of facial nerve excised. A silk suture, no. 000000, fixed graft to divided ends of facial nerve above and below. Upper suture was difficult to place, as facial nerve beyond bend was soft and small. As it was not clear whether this suture was torn out of facial nerve or not, two more grafts from intercostohumeral nerve were laid on Bell's nerve between divided ends of facial nerve. They lay in good position, their extremities touching cut ends of facial nerve. Gold leaf was not used.

June 2 (64 days): Monkey anesthetized. No winking of right eye. Faradic Current: Good response in muscles around mouth; less in lower lip than in upper. None in orbicularis palpebrarum.

July 7 (99 days): Mouth symmetrical. Face appeared normal. Winking synchronous but not as complete on right side as on left. Corneal reflex present in right eye. Spasm of muscles above angle of mouth and in lower lip occurred synchronously with effort to wink. (It was noted later that spasm went on during sleep and during anesthesia.) Monkey anesthetized. Faradic Current: Fine response in upper and lower lips; less response in orbicularis palpebrarum. Galvanic Current (6 ma., anodal closure): Same response on both sides.

EXPERIMENT 31.—Rhesus monkey. Operation (right side) on March 2, 1931: Great auricular-facial end-to-end anastomosis. Facial nerve was divided at stylomastoid foramen. Great auricular nerve was found on sternomastoid muscle and divided at level of lower border of mastoid process. Suture of ends of nerves was effected with silk no. 000000 without tension. A piece of gold foil, $\frac{1}{2.500}$ inch in thickness, was placed on inner and outer aspects of anastomosis.

Facial nerve removed from aqueduct of Fallopius: About three fifths of an inch (1.5 cm.) of nerve was exposed by removing outer boundary of canal in region of stylomastoid foramen. On gently pulling on stump of facial, 3 cm. of nerve was drawn out and cerebrospinal fluid escaped. The nerve had probably been avulsed from lower border of pons varolii.

June 1: Monkey anesthetized (90 days). Faradic Current: Definite response in orbicularis oris; slight response in orbicularis palpebrarum.

July 4: Monkey anesthetized (124 days). Faradic Current: Marked response in orbicularis oris when electrode was placed at angle of mouth; also in levator nasi; faint twitch in levator labii superioris. Galvanic Current (6 ma.): Good response in all muscles with both positive and negative closure, but more with negative.

EXPERIMENT 32.—Rhesus monkey. Operation (right side, on March 4, 1931): Transverse cervical-facial end-to-end anastomosis. Facial nerve divided at systomastoid foramen. Transverse cervical or superficial cervical nerve was found on middle of sternomastoid muscle and divided beyond anterior border of muscle. This gave enough length of nerve to enable anastomosis to be made without tension. Around anastomosis was coiled gold foil, $\frac{1}{2.500}$ inch in thickness. Facial nerve

removed from fallopian canal: Mastoid process had been opened at a previous operation. On freeing sheath of nerve from its attachment at stylomastoid foramen and gently pulling on the stump of the nerve, about 2 cm. of nerve was drawn out of its canal. This was followed by cerebrospinal fluid which quickly filled mastoid cavity. A piece of muscle was cut from the sternomastoid and placed in mastoid cavity. Flow of cerebrospinal fluid ceased at once.

June 5: Monkey anesthetized (88 days). Faradic Current: Definite response in orbicularis oris and in levator nasi. Slight response in orbicularis palpebrarum. No winking of right eye.

July 3: Monkey anesthetized (116 days). Faradic Current: Definite response in levator labii superioris alaeque nasi. Also response in all muscles of upper and lower lips. No movement obtained in orbicularis palpebrarum. No movement of muscles of face occurred when electrode was placed over region of trunk of facial nerve. Galvanic Current (6 ma., anodal closure): Larger and more sluggish movements on right side than on left. Upper muscle contractions quicker and smaller.

EXPERIMENT 33.—Rhesus monkey. Operation (right side) on March 5, 1931: Great occipital-facial end-to-end anastomosis. Great occipital nerve was found by a vertical incision to right of midline behind. It was followed a short distance upward underneath scalp and followed downward through trapezium and complexus. Nerve was divided just above superior curved line and turned forward. A gold tube made of gold foil, $\frac{1}{2.500}$ inch in thickness and 2 cm. in length, was threaded on nerve. Facial nerve was divided at stylomastoid foramen. Divided ends of two nerves were brought together without tension and sutured with catgut no. 0000. Gold tube reached to anastomosis. A piece of sternomastoid muscle lay on inner side of anastomosis; at close of operation, parotid gland fitted down over it. Facial nerve removed from fallopian canal: Stylomastoid foramen was opened up, and nerve at this spot was freed from its attachments to bone. Gentle traction was then applied to stump of nerve. About 15 cm. came away, and cerebrospinal fluid escaped. A piece of sternomastoid muscle placed in partially opened mastoid cavity stopped escape of cerebrospinal fluid.

June 1: Monkey anesthetized (87 days). Gold tube could be felt beneath skin. Faradic Current: Slight response in orbicularis oris; with a strong current there was a good response.

July 7 (123 days): In right eye no protective reflex or corneal reflex. Slight drooping of right side of lower lip. No spasmodic movement of muscle seen. Monkey anesthetized. Faradic Current: Fair response in lower lip; slight response in upper lip. Galvanic Current (6 ma., anodal and cathodal closure): Equal response on the two sides.

EXPERIMENT 34.—Rhesus monkey. Operation (right side) on April 8, 1931: Auriculotemporal-facial end-to-end anastomosis. Auriculotemporal nerve was found under attrahens aurem muscle; it was divided at upper border of this muscle and followed downward till it passed forward under neck of condyle of jaw. Facial nerve was divided at stylomastoid foramen. An incision in parotid gland was required in order to bring ends of two nerves into apposition. Suture of nerves was effected in this incision in parotid with silk no. 000000 and with very little tension. Mastoid process had been in part excavated by a previous operation. Facial nerve was avulsed as far as a little beyond the bend.

June 1: Monkey anesthetized (54 days). Faradic Current: Definite response in orbicularis oris.

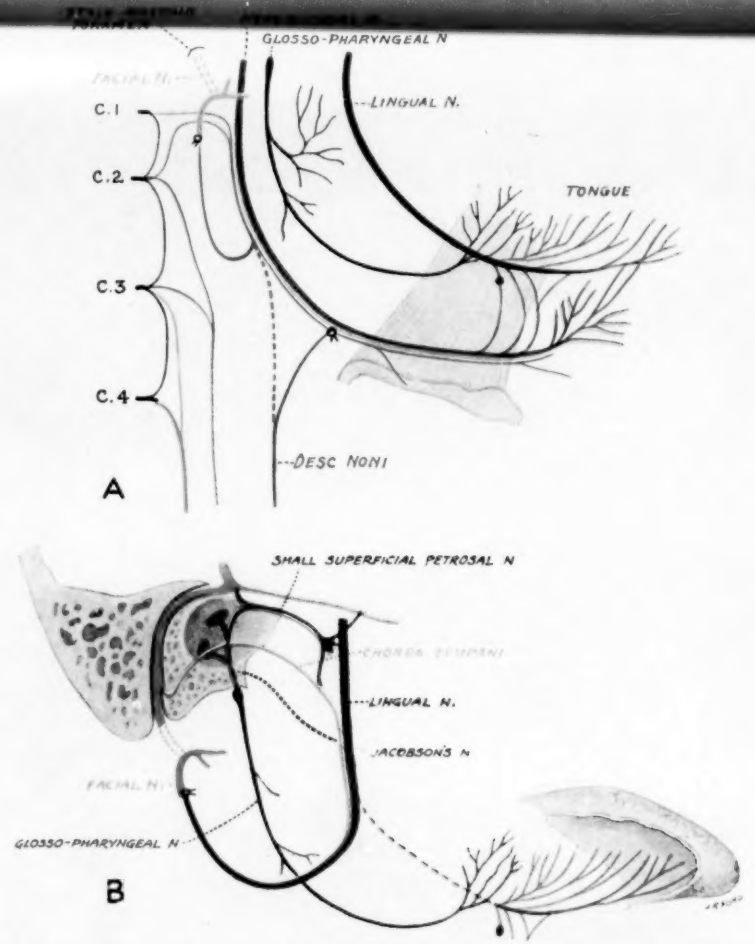


Fig. 2.—*A*, descendens noni-facial end-to-end anastomosis; hypoglossal-descendens noni side-to-end anastomosis. *B*, facial-lingual-chorda tympani anastomosis. The small superficial petrosal nerve from Jacobson's nerve and the tympanic plexus communicate with the chorda tympani.

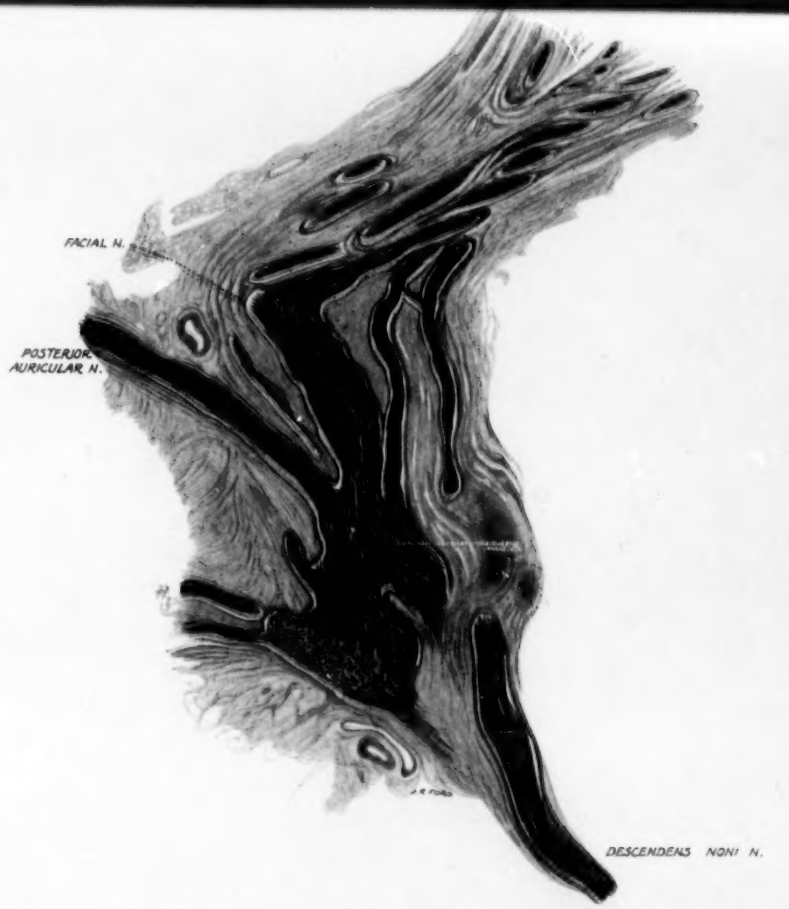


Fig. 3.—Microscopic drawing (low power) of an end-to-end anastomosis between the descendens noni and facial nerves. The specimen was obtained fourteen months after the experiment was done. Stroebe stain.

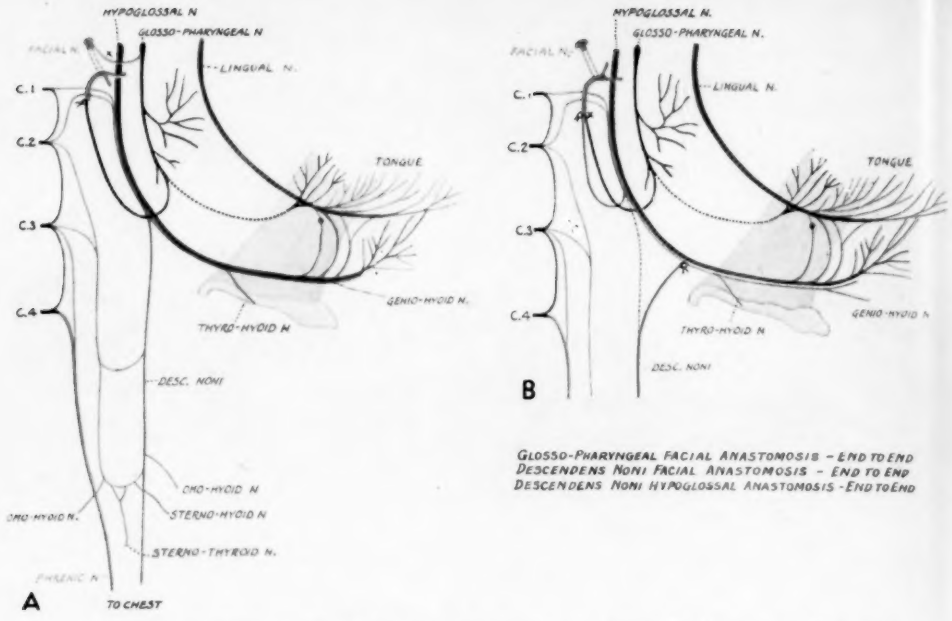


Fig. 4.—*A*, glossopharyngeal-facial anastomosis, end to end (*X*, ansa of Haller). *B*, glossopharyngeal-facial end-to-end anastomosis; descendens noni-facial end-to-end anastomosis; descendens noni-hypoglossal end-to-end anastomosis.

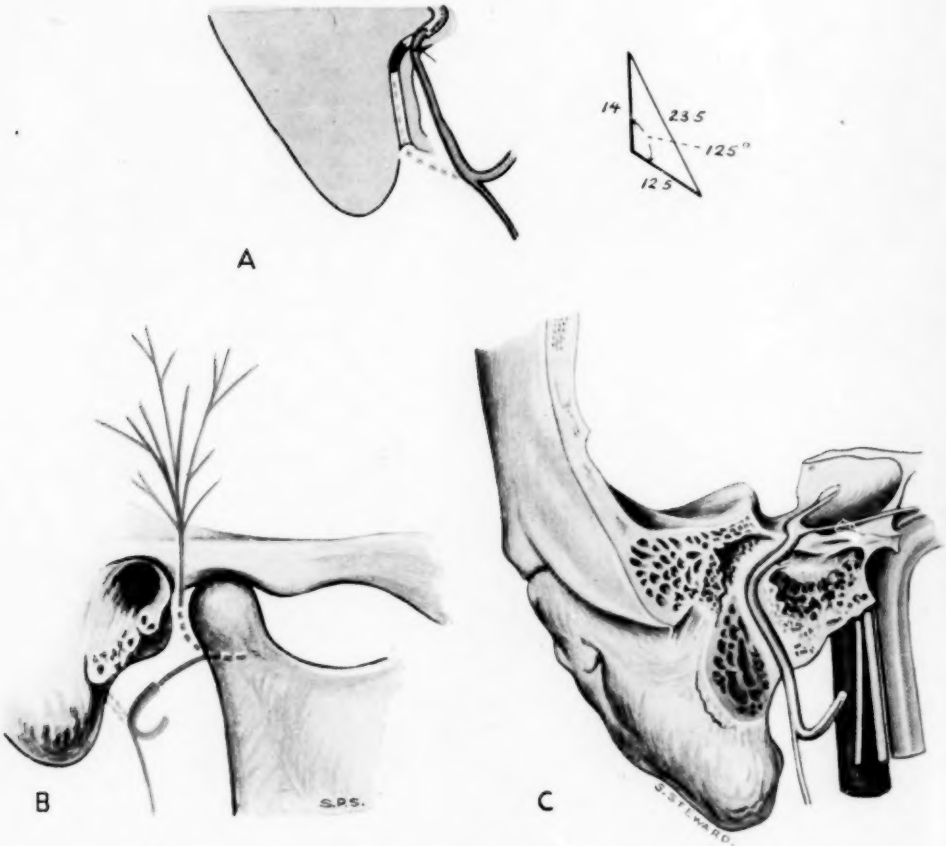
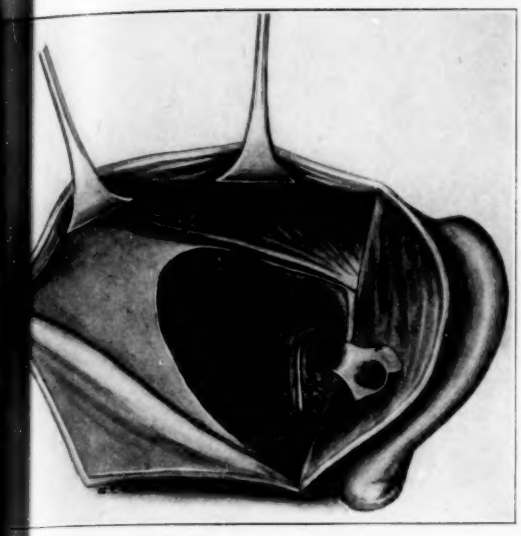


Fig. 5.—*A*, injury of the facial nerve at the bend. Displacement of the facial nerve from the canal allowing an end-to-end suture to be done. *B*, auriculotemporal-facial anastomosis. *C*, facial nerve dissection. Antero-external view at right angles to the antral-tympanal-eustachian axis. This axis forms an angle of 45 degrees with the anteroposterior median plane.



6.—Facial nerve dissection. View at right angles sagittal plane.

Fig. 8.—Dissection of axilla to show Bell's nerve. Suture reuniting the divided ends after the removal of a piece 5 mm. in length for use as a graft. (Modified from Poirier and Charpy, after Hirschfeld.)

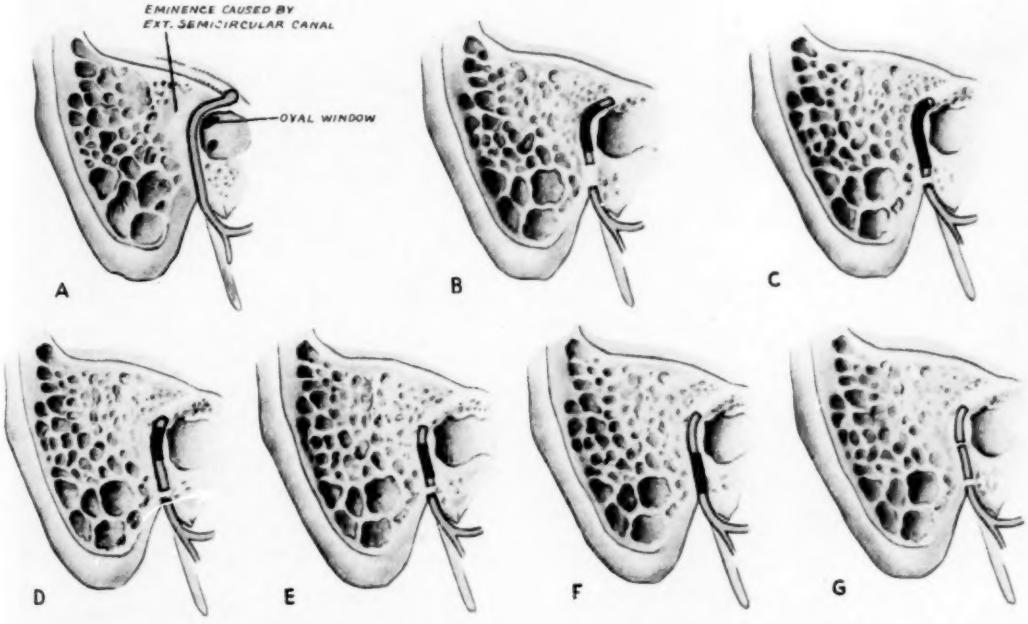


Fig. 7.—*A*, outer wall of fallopian canal removed from stylomastoid foramen to region of geniculate ganglion. *B*, a graft of 10 mm. of Bell's nerve, not reversed. *C*, a graft of 12 mm. of the facial nerve, reversed. *D*, a graft of 5 mm. of the facial nerve, reversed. *E*, a graft of 6 mm. of the sixth intercostal nerve, not reversed. *F*, a graft of 7 mm. of the descendens noni, not reversed. *G*, facial nerve divided and left in the canal.

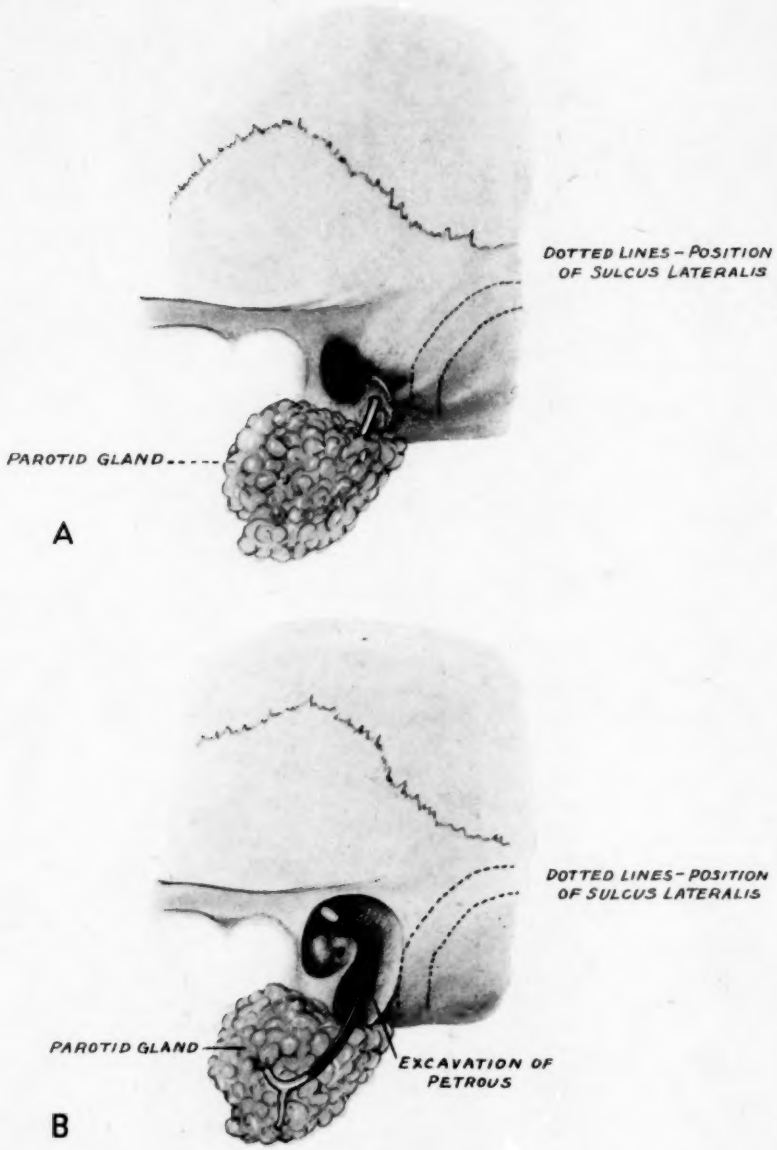


Fig. 9.—*A*, temporal bone and exposed facial nerve at the age of 8 months. *B*, 27 mm. nerve graft (case 1).

Fig. 10.—*A*, photograph taken a few days after a nerve grafting operation at the Manhattan Hospital for facial palsy on March 18, 1931 (case 1). *B*, photograph taken on May 18, 1931, sixty-one days after the operation. *C*, photograph taken on June 28, 1931, 101 days after the operation.

Fig. 11.—Fracture of the middle ɔssa of the base of the skull. The fracture crosses the aqueduct of Fallopius at the bend.

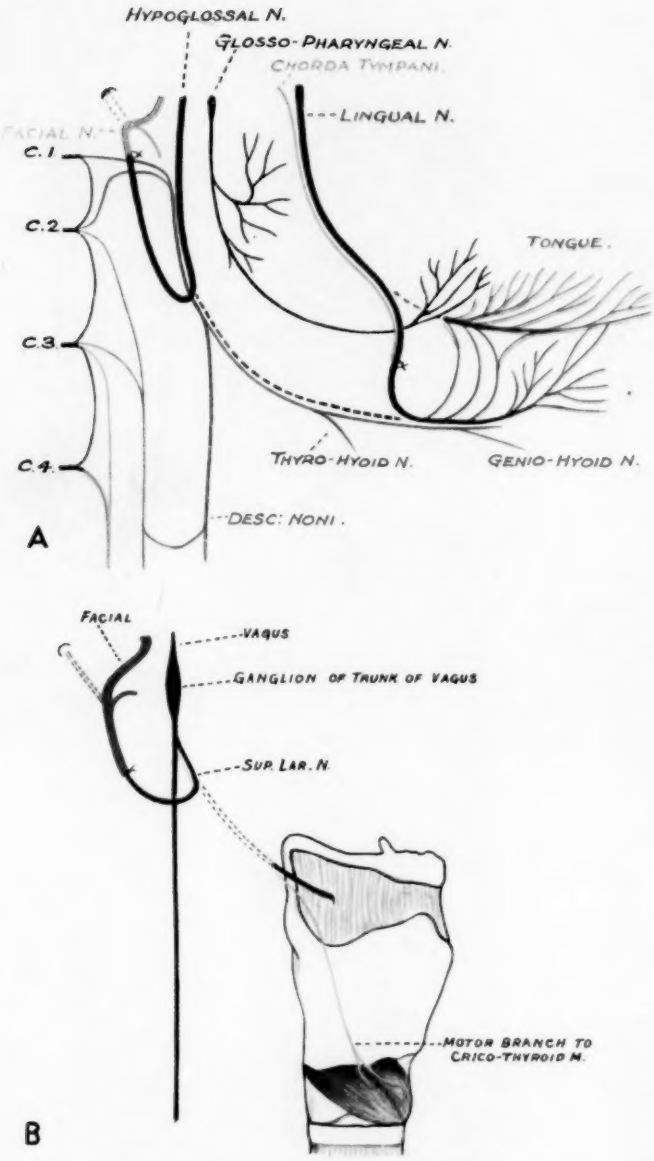


Fig. 12.—*A*, hypoglossal-facial anastomosis; lingual-facial anastomosis. *B*, superior laryngeal-facial end-to-end anastomosis.

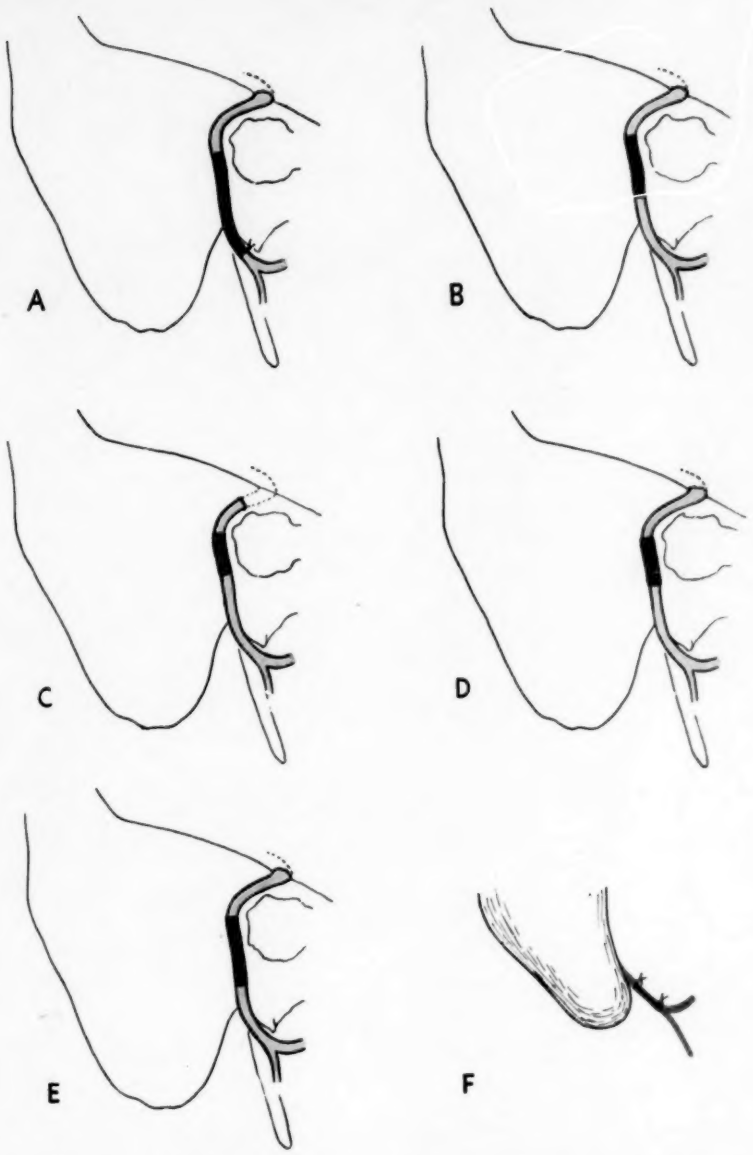


Fig. 13.—Canal opened from the region of the geniculate ganglion to the stylomastoid foramen. A 16 mm. graft of Bell's nerve was introduced. *B*, canal opened from the region of the geniculate ganglion to the stylomastoid foramen. A 9 mm. graft was introduced; no sutures were used. *C*, canal opened 3 mm. beyond the bend to the stylomastoid foramen. A 6 mm. graft was introduced; no sutures were used. *D*, canal opened from the region of the geniculate ganglion to the stylomastoid foramen; two 7 mm. grafts were introduced; no sutures were used. *E*, canal opened from the region of the geniculate ganglion to the stylomastoid foramen. A 10 mm. graft was introduced; there were no sutures. *F*, mastoid process of a rhesus monkey; 5 mm. of the trunk of the facial nerve was replaced by 5 mm. from Bell's nerve.

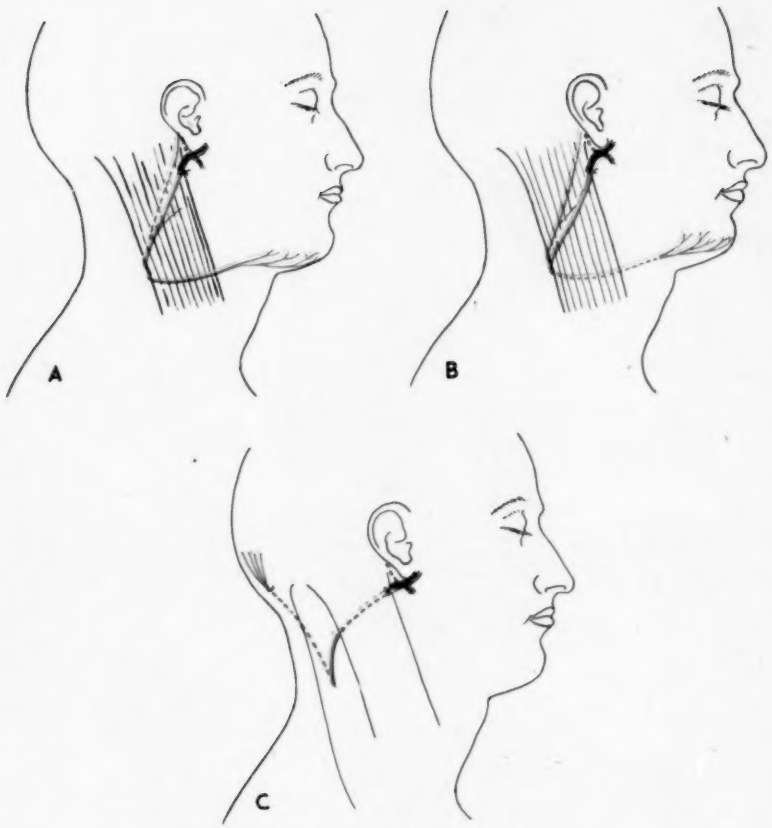


Fig. 14.—*A*, great auricular-facial anastomosis. *B*, transverse cervical-facial anastomosis. *C*, great occipital-facial anastomosis.

July 7 (90 days): No corneal reflex. Right lower lip drooped a little. Faradic Current: Definite response in orbicularis oris. Galvanic Current (6 ma.): Cathodal closure gave more active response than anodal.

EXPERIMENT 35.—Rhesus monkey. Operation (right side) on March 28, 1931: Trunk of facial nerve replaced by a length of Bell's nerve. Trunk of facial nerve was exposed from stylomastoid foramen to the point where it divided into its two main branches. Ten millimeters of trunk of facial nerve replaced by 10 mm. of Bell's nerve. Suturing was done with no. 000000 silk and a three-eighths inch special needle. Nerve graft was loosely surrounded with a tube of gold foil, $\frac{1}{2.500}$ inch in thickness. Cut ends of Bell's nerve in axilla were brought together with a catgut suture no. 00000.

May 17: Monkey anesthetized (50 days). Faradic Current: Definite response in upper lip muscles and in orbicularis oris.

June 2: Monkey anesthetized (66 days). Faradic Current: Good contraction of upper lip muscles; less response in lower lip muscles.

July 7 (101 days): Mouth symmetrical; no sign of palsy. Right corneal reflex not as strong as left. Right nostril not as open as left; it moved slightly forward and backward during breathing. Spontaneous twitch of muscles around mouth. This was synchronous with winking, and a protective wink increased the movement. On touching cornea, spasmodic movement of upper lip was increased with increase of contraction of orbicularis palpebrarum. Monkey anesthetized. Faradic Current: Excellent response in upper lip; less response in lower. With electrode over region of trunk of facial nerve upper lip was drawn slightly backward and upward. Galvanic Current (6 ma., anodal closure): Response equal on both sides.

EXPERIMENT 36.—Rhesus monkey. Operation (right side) on March 20, 1931: Ten millimeters of Bell's nerve replaced by 10 mm. of descendens noni nerve; 10 mm. of descendens noni nerve replaced by 10 mm. of Bell's nerve.

July 20: Monkey anesthetized (122 days): Faradic Current: In incision in neck, stimulation of hypoglossal above origin of descendens noni caused contraction of sternohyoid muscle. In incision in axilla, stimulation of Bell's nerve above graft region caused contraction of all serrations of serratus magnus.

EXPERIMENT 37.—Rhesus monkey. Operation (right side) on March 15, 1931: Thirteen millimeters of Bell's nerve replaced by 13 mm. of internal cutaneous nerve taken from in front of elbow; 13 mm. of internal cutaneous nerve replaced by 13 mm. of Bell's nerve. Nerve grafts were not reversed.

July 20 (127 days): Faradic Current: In incision in axilla, stimulation of Bell's nerve above graft caused contraction of all serrations of the serratus magnus. Examination of skin of forearm for anesthesia below graft operation area was not satisfactory.

EXPERIMENT 38.—Rhesus monkey. Operation (right side) on March 13, 1931: Six millimeters of Bell's nerve in axilla excised, reversed and sutured into position again. Suture, catgut no. 0000.

June 2: Monkey anesthetized (81 days). Faradic Current: Through skin, contraction of right serratus less than that of left.

July 21: Monkey anesthetized (130 days). Faradic Current: In incision in axilla, electrode placed on nerve proximal to graft caused full contraction of all serrations of serratus magnus.

EXPERIMENT 39.—Rhesus monkey. Operation on March 1, 1931: Five millimeters of Bell's nerve used to replace 5 mm. of trunk of facial nerve. Suture,

black silk no. 000000. Anastomosis not surrounded by extraneous matter. Trunk of facial nerve was one-half inch in length. When nerve graft had been fixed in position, ends of Bell's nerve in axilla were brought together by suture.

April 17: Monkey anesthetized (47 days). Right eye lagged behind left in winking; otherwise face was symmetrical. Farradic Current: Good contraction of upper lip muscles. With stronger current, orbicularis oris and lower lip muscles contracted; slight response in orbicularis palpebrarum and in occipitofrontalis.

June 19: Animal escaped and was shot a week later. No further clinical record was available.

Fig. 15.—Reproductions of drawings by Sir Charles Bell taken from his work entitled "The Anatomy and Philosophy of Expression" (1872). *A*, laughter. *B*, weeping. *C*, fear. *D*, pain. *E*, jealousy. *F*, remorse. *G*, madness. The drawings, depicting the expression of the emotions in man, may be compared with the photographs of the faces of baboons (figs. 16 to 20). The baboon does not smile or laugh. He grunts satisfaction when pleased with food given him. He exhibits the emotions of fear and surprise with elevation of the eyebrows; also the emotion of anger. Sometimes a kind of pleasure or affection is shown when the head or back is stroked or tickled, the lips opening and closing rapidly with a smacking sound. Yawning is common among baboons, especially in the afternoon. As in man, the jaws are widely separated; the risorius and other muscles pull back the angles of the mouth and the eyelids close. A few months after the performance of a nerve graft operation in the aqueduct of Fallopius there appear to be three stages in the act of yawning: 1. The right side of the mouth (the side of operation) is pulled on more strongly than the left side. 2. Then the muscles of the left side of the mouth show greater strength and pull the mouth to the left. 3. Lastly, as the yawn reaches its maximum, the right side of the face is again pulled on more strongly than the left side. It is clear that the neokinetic centers of the two cerebral hemispheres are not yet in harmonious cooperation.

FINAL EXAMINATIONS, CLINICAL AND PHYSIOLOGIC

It appears reasonable to conclude:

1. The plan adopted in the anastomosis experiments, to prevent the severed ends of the facial nerve from reuniting, was successful.

2. When the usual path of a nervous impulse, between the nerve cells in the brain and the motor end-organ in the muscle, is not available, it can and does travel by another route.

3. In some of the experiments there is an indication of how soon after the substitution of a nerve graft for a portion of a nerve, the function of the nerve may be restored. This restoration depends on the length of the nerve graft, the age of the patient and perhaps on other factors that are not so well known.

EXPERIMENT 1.—Baboon. Operation on Aug. 21, 1930: Hypoglossal-facial and lingual-hypoglossal anastomosis (right side). Final examination on Aug. 1, 1931. Monkey anesthetized. Faradic Current: Response equal on both sides of face. Muscles of right side of face had completely recovered. On left side, electrode was placed over region of trunk of facial nerve. Response in all muscles of left side of face. On right side, electrode was pushed deeply into neck below posterior part of lower edge of body of lower jaw. Response in all muscles of right side of face. Tongue: No sign of atrophy in right side of tongue. Anterior half was plump like left side, but surface was smooth and red, which contrasted with corresponding surface on left side.

Slight but definite response to faradic current in anterior half of right side of tongue. Galvanic Current (6 ma., anodal closure): Muscle contractions had a wider excursion in anterior half of right side of tongue than in corresponding region on left side. No response to electrical stimulation in posterior half of right side of tongue. At this moment, baboon died, so stimulation of rolandic cortex was not attemped.

Brain removed, and nerves stimulated by the faradic current in posterior fossa of base of skull.[78] Right Facial Nerve: No response in muscles of right side of face. Right Hypoglossal Nerve. Contraction of muscles of right side of face. Left Facial Nerve: Contraction of muscles of left side of face. Left Hypoglossal Nerve: Contraction of muscles of left side of tongue.

EXPERIMENT 2.—Baboon. Operation on Aug. 22, 1930: Descendens nonifacial and hypoglossal-descendens noni anastomosis (right side). Final examination on Aug. 1, 1931. Monkey anesthetized. Faradic Current: Muscles of face on both sides responded equally. Exposure of right and left rolandic cortex. On stimulation of left and right facial areas, equal response in muscles of opposite side of face. On stimulation of right cortex, representation of the movements of muscles of left side of face and tongue were more mixed, and less separated than when corresponding area was stimulated on left side.

Brain removed, and nerves in posterior fossa of base of skull stimulated. Right Facial Nerve: No response. Left Facial Nerve: All muscles of face contracted. Notes do not reveal results of stimulation of hypoglossal nerves; however, as descendens noni comes from first and second cervical nerves, and not from cells of hypoglossal nucleus, no movement of right facial muscles would have

78. In stimulation of the cortex cerebri or the nerves in the posterior fossa of the base of the skull, the Sherrington single electrode was always employed.

occurred if right hypoglossal had been stimulated in posterior fossa. Dissection of Neck: On exposure, sternohyoid and sternothyroid muscles of both sides appeared exactly alike. On direct electrical stimulation of muscles, contractions on right side equaled those on left.

EXPERIMENT 3.—Baboon. Operation on Aug. 12, 1930: Glossopharyngeal-facial end-to-end anastomosis. This monkey is being reserved for further observation.

EXPERIMENT 4.—Baboon. Operation on Aug. 13, 1930: Glossopharyngeal-facial end-to-end anastomosis (right side). Final examination on Aug. 4, 1931. Monkey anesthetized. Faradic Current: Complete recovery of all muscles on right side of face. Slight spasmodic twitch noted at angle of mouth, involving both upper and lower lips. This was not associated with winking, and was not present under deep anesthesia. When electrode was placed under angle of jaw on right side, all muscles of right side of face contracted. On left side same result was obtained by placing electrode over stylomastoid foramen or a little in front of it. Exposure of the left rolandic cortex. On faradic stimulation of facial area, all muscles of right side of face contracted.

Brain removed and nerves stimulated in posterior fossa. Right Facial Nerve: No response. Right Glossopharyngeal Nerve: All muscles of right side of face contracted.

EXPERIMENTS 5 and 17.—Baboon. Operation on Aug. 14, 1930: Glossopharyngeal-facial end-to-end anastomosis (right side). Operation on March 8, 1931: Graft of Bell's nerve replaced portion of facial nerve in aqueduct of Fallopius (left side). Final examination on July 24, 1931. Before anesthesia, face looked normal. Protective wink on both sides; a little quicker on left than on right. Monkey anesthetized. Eyelids equally closed. Corneal reflex on both sides as animal came out of deep anesthesia. On right side frequent spasmodic twitches of upper and lower eyelids, levator menti and risorius (retraction of angle of mouth). These spasmodic movements were synchronous with spasmodic contractions of posterior half of right side of tongue. No spasmodic movement of left side of face. Faradic Current: All muscles on both sides of face responded, but response was better on left side than on right. Galvanic Current (6 ma., anodal closure): On left side, quick small normal movements. On right side, slower and wider excursion contractions. Stimulation of Right and Left Rolandic Cortex; Left Facial Area: No movement of right facial muscles. On stimulation just above facial area, right thumb and finger movements active; on stimulation below, contraction of muscles of right side of tongue. Facial area seemed dead on faradic stimulation. Right Facial Area: Good contraction of all left facial muscles.

Brain removed and nerves in posterior fossa of base of skull stimulated. Left Facial Nerve: Contraction of all muscles of left side of face. Right Facial Nerve: No contraction of right facial muscles. Right Glossopharyngeal Nerve: No contraction of right facial muscles.

EXPERIMENT 6.—Baboon. Operation on Aug. 15, 1930: Glossopharyngeal-facial end-to-end anastomosis (right side). Final examination on Aug. 6, 1931. Right eyelids never completely closed. Protective wink (right side) only a flicker. Monkey anesthetized. On right side, no corneal reflex. Lower lip drooped a little. Occasional spontaneous retraction of angle of mouth (risorius). Faradic Current: Poor response in all muscles of upper and lower lips, including orbicularis oris. Orbicularis palpebrarum gave no response. Stronger current over anastomosis in

neck caused marked retraction of angle of mouth. Stimulation of Right and Left Rolandic Cortex; Left Facial Area: Small contractions of muscles of right side of upper and lower lips. Marked retraction of angle of mouth. Right Facial Area: Full contractions of all muscles of left side of face.

Brain removed, and nerves stimulated in posterior fossa of base of skull. Right Facial Nerve: No response. Right Glossopharyngeal Nerve: Marked retraction of mouth and slight movements of muscles of upper and lower lips. Left Facial Nerve: Contraction of all muscles of right side of face.

EXPERIMENT 7.—Baboon. Operation on Aug. 16, 1930: Glossopharyngeal-facial end-to-end anastomosis (right side). Final examination on Aug. 7, 1931. Monkey anesthetized. Corneal reflexes equal. Eyeballs equally exposed. Frequent spontaneous spasm of upper and lower lip muscles, and retraction of angle of mouth; these movements were synchronous with winking. They occurred around the angle of the mouth without winking movement, but appeared to be increased when associated with winking. Faradic and Galvanic Current: Response of facial muscles equal on both sides. Stimulation of Left Rolandic Cortex; Left Facial Area: Contraction of all muscles of right side of face; response normal and complete.

Brain removed and nerves stimulated in posterior fossa of base of skull. Right Facial Nerve: No response. Left Facial Nerve: All facial muscles contracted strongly. Right Glossopharyngeal Nerve: Good contraction of muscles of upper and lower lips and marked retraction of angle of mouth. With stronger current, right eyelids closed.

EXPERIMENT 8.—Baboon. Operation on Aug. 17, 1930: Glossopharyngeal-facial end-to-end anastomosis (right side). Final examination on Aug. 9, 1931. Monkey anesthetized. On right side of face, spontaneous spasm of risorius and muscles of upper and lower lips, not always associated with winking of right eyelids. These movements were not observed except under partial anesthesia. In posterior third of right side of tongue, tonic spasm observed, causing a deep sulcus to appear on surface. This contraction of muscles of tongue was not certainly seen to be synchronous with facial spasm. Faradic and Galvanic Current: Completely equal response on both sides of face. Stimulation of Left Rolandic Cortex; Facial Area: Normal response in muscles of right side of face.

Brain removed and nerves in posterior fossa of base of skull stimulated. Right Facial Nerve: No response. Right Glossopharyngeal Nerve: Good contraction of muscles of upper and lower lips and marked retraction of angle of mouth. Dissection of Neck: Stimulation of glossopharyngeal nerve at lower border of jugular foramen (petrous ganglion exposed) caused contraction of all muscles of face.

EXPERIMENT 9.—Baboon. Operation on Aug. 28, 1930: Lingual-facial end-to-end anastomosis (right side). Final examination on July 26, 1931. Face looked symmetrical at rest, but right upper canine was slightly exposed. Monkey anesthetized. No right corneal reflex. Eyes winked synchronously. Right eyelids closed more completely than left. When left cornea was touched, right upper eyelid closed over cornea at once. Faradic Current: All upper lip and lower lip muscles responded, but response was not strong. No response in orbicularis palpebrarum or in occipitofrontalis. Galvanic Current (6 ma., anodal closure): Wider and slower contraction excursions of muscles of right side as compared with those of left side. At this moment, monkey died; hence no attempt was made to stimulate the cortex cerebri.

Brain removed and nerves in posterior fossa base of skull stimulated. Right Facial Nerve: No response. Right Fifth Nerve: No response in masticatory muscles, but muscles of right side of upper lip contracted, and angle of mouth was drawn backward. Left Facial Nerve: Contraction of all muscles of left side of face. Left Fifth Nerve: Contraction of masticatory (temporal, masseter, etc.) muscles. Dissection of Nerves of Face (facial branches): These appeared normal, and on electrical stimulation caused contraction of corresponding facial muscles.

EXPERIMENT 10.—Baboon. Operation on Aug. 23, 1930: Auriculotemporal-facial end-to-end anastomosis (right side). Final examination on Aug. 3, 1931. Monkey anesthetized. Spontaneous twitch of muscles of right side of upper and lower lips occurred, which was synchronous with winking spasm. Those spasmodic movements did not take place except under partial anesthesia. Faradic and Galvanic Current: Response equal on both sides in all muscles. Stimulation of Left Rolandic Area of Cortex; Left Facial Area: All muscles of right side of face contracted normally.

Brain removed and nerves stimulated in posterior fossa of base of skull. Right Facial Nerve: No response. Left Facial Nerve: Contraction of all muscles of left side of face. Right Fifth Nerve: Contraction of all muscles of right side of face. Left Fifth Nerve: Contraction of muscles of mastication.

EXPERIMENT 11.—Baboon. Operation on Aug. 11, 1930: Superior laryngeal-facial end-to-end anastomosis (right side). Final examination on Aug. 2, 1931. Monkey anesthetized. No corneal reflex (right eye). Vocal chords moved equally in tranquil respiration, but right chord was slack and not tense and straight like left chord. On electrical stimulation through skin of neck, chords came together with a snap. Faradic Current: Good response in lower lip muscles. Poor response in upper lip muscles. Levator nasi contracted, as did orbicularis oris; angle of mouth retracted by risorius. Response in orbicularis palpebrarum only a flicker. Galvanic Current (6 ma., anodal closure): Response of muscles of upper and lower lips equal on both side. Faradic Current (strong): Electrode pushed in below middle of right lower jaw (with idea of stimulating anastomosis). Good contraction of lower lip muscles and risorius. Electrode placed in front of right stylomastoid foramen. Backward and forward movement of pinna, and slight retraction of angle of mouth. Stimulation of Rolandic Cortex on Both Sides of Brain; Left Facial Area: No movement of muscles of right side of face, though stimulation above thumb and fingers areas and below tongue area gave normal movements of these organs. Right Facial Area: Normal movements of all muscles of face.

Brain removed and nerves in the posterior fossa of base of skull stimulated. Right Facial Nerve: No response. Left Facial Nerve: Full response. Right Vagus Nerve: Contraction of muscles of right side of lower lip and retraction of angle of mouth.

EXPERIMENT 12.—Baboon. Operation on Aug. 27, 1930. Facial nerve in aqueduct divided in two places, 12 mm. apart and reversed. Monkey was found dead on June 17, 1931, so that no further clinical examinations could be made.

EXPERIMENT 13.—Mandrill baboon. Operation on Aug. 27, 1930: Facial nerve divided in aqueduct in two places, 5 mm. apart and reversed. This monkey is being reserved for further observation.

EXPERIMENT 14.—Baboon. Operation on Sept. 17, 1930: Six millimeters of facial nerve removed from aqueduct and replaced by 6 mm. of intercostal nerve. This monkey is being reserved for further observation.

EXPERIMENT 15.—Baboon. Operation on Sept. 16, 1930: Portion of facial nerve in aqueduct, 7 mm. in length, was removed and replaced by 7 mm. of descendens noni nerve (right side). Final examination on Aug. 7, 1931, showed perfect recovery of right side of face. Monkey anesthetized. No spontaneous spasm. Faradic and Galvanic Currents: Equal response on both sides of face. Faradic current electrode placed on skin over trunk of facial nerve (right). Good response in right facial muscles. Stimulation of Left Rolandic Cortex; Facial Area: All muscles of right side of face contracted.

Brain removed and nerves stimulated in posterior fossa. Right and Left Facial Nerves: Equal and full contraction of muscles of face.

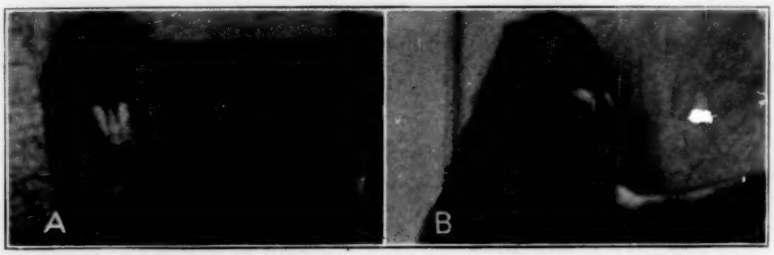


Fig. 16 (experiment 13).—Photographs nine months after operation. *A*, face in repose, showing complete recovery. *B*, right side (side of operation) drawing up in beginning grimace.

Fig. 17 (experiment 14).—Photograph about nine months after operation (right side). The baboon is surprised; he raises the eyebrows, opens the eyes widely, raises the upper lip and tip of the nose and draws back the corners of the mouth. All movements are equal on the right and left sides.

EXPERIMENT 16.—Baboon. Operation on Sept. 19, 1930: Facial nerve divided in aqueduct and left to unite in canal (right side). Final examination on Aug 8, 1931. Monkey anesthetized. Slight spontaneous spasm in risorius and in levator labii superioris alaeque nasi. Faradic and Galvanic Currents: Equal and normal response on right and left sides of face. Stimulation of Left Rolandic Cortex; Facial Area: Good response in all muscles of right side of face.

Brain removed and nerves stimulated in posterior fossa. Right Facial Nerve: All muscles of right side of face contracted. (On stimulation of right glosso-

pharyngeal nerve, right wall of pharynx moved upward and outward [Stylopharyngeus]. Soft palate and uvula moved to right, and extreme posterior part of right side of tongue moved slightly in same direction.)

EXPERIMENT 18.—Baboon. Operation on April 4, 1931: Sixteen millimeters of Bell's nerve substituted for 16 mm. of facial nerve in aqueduct (right side). Final examination on Aug. 8, 1931 (126 days). During yawning, right angle of mouth was first pulled on more than left; then muscles on left side pulled mouth

Fig. 18 (experiment 16).—Photograph about eight months after operation, at the completion of a yawn, showing symmetrical contraction of the facial muscles. The risorius fully drawn back. Levator labii superioris alaeque nasi lifts the upper lip and nose; the eye closes.

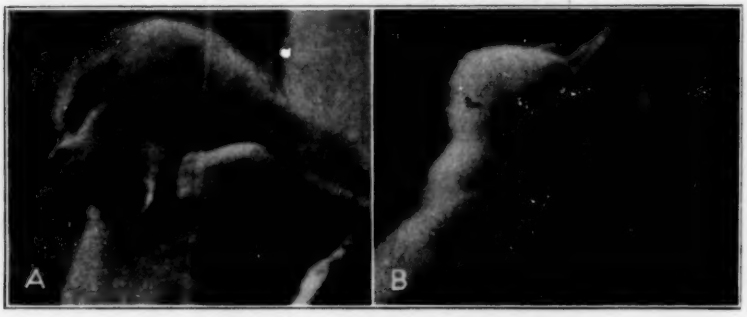


Fig. 19 (experiment 19).—Photographs about nine weeks after operation. *A*, beginning of a yawn. The risorius pulls the right angle of the mouth a little farther back than the left; the upper lip and nose are still down. *B*, end of the yawn. The risorius pulls the angles of the mouth back nearly equally on the right and left sides. The nose and upper lip are lifted evenly on both sides; the eyes are closed equally.

somewhat to the left; lastly, just before completion of yawn, muscles of right side pulled mouth to the right. Monkey anesthetized. Faradic Current: Good response in risorius, but response was poor in muscles of upper and lower lips and in orbicularis oris. No response in orbicularis palpebrarum. Right corneal reflex was brisk. Galvanic Current (10 ma., anodal closure): Equal response in muscles

of both sides of face. Stimulation of the Rolandic Cortex of Both Hemispheres; Left Facial Area: Definite but slight contraction of right side of upper and lower lip. Right Facial Area: Normal full response on left side of face.

Brain removed and nerves in posterior fossa exposed. Right Facial Nerve: All muscles on right side of face contracted.

EXPERIMENT 19.—Baboon. Operation on April 4, 1931: A 9 mm. graft of Bell's nerve substituted for 9 mm. of facial nerve in aqueduct. This monkey is being reserved for further observation.

EXPERIMENT 20.—Baboon. Operation on April 5, 1931: A 6 mm. graft of Bell's nerve substituted for 6 mm. of facial nerve in aqueduct (right side). Final examination on Aug. 10, 1931 (127 days). Animal yawned symmetrically. Good protective wink (right). Monkey anesthetized. Spontaneous twitch of risorius, involving muscles of upper and lower lips. These spasmodic movements were not associated with winking. Faradic Current: Response in all muscles on right side, but contractions were not so wide, as on left side of face. Risorius did not respond.

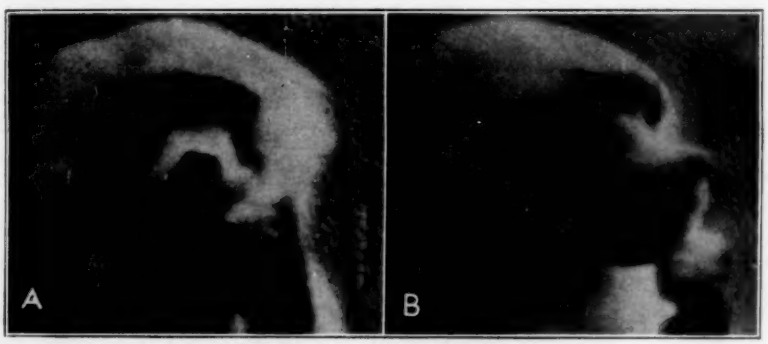


Fig. 20 (experiment 21).—Extracts from moving picture about twelve weeks after operation. *A*, the right food pouch is full; left is just being emptied with the aid of the hand. *B*, the right food pouch has been emptied by the action of the facial muscles alone; the hand is being withdrawn, just after emptying the left food pouch.

Galvanic Current (6 ma., anodal closure): Response the same on both sides of face. Stimulation of Rolandic Cortex of Both Hemispheres; Left Facial Area: Contraction of all muscles of right side of face. Right Facial Area: Contractions of left facial muscles were wider in range than those on right side.

Brain removed and nerves stimulated in posterior fossa. Right Facial Nerve: All muscles contracted. Left Facial Nerve: Contractions on this side were wider and stronger than those on right. Dissection of Axilla: Faradic stimulation of Bell's nerve proximal to the suture produced contraction of all serrations of serratus magnus.

EXPERIMENT 21.—Baboon. Operation on April 6, 1931: Two lengths of Bell's nerve (7 mm. each) substituted for 7 mm. of facial nerve in aqueduct (right side). Final examination on August 10 (126 days). In wide yawning, the two sides of mouth moved equally. Monkey anesthetized. Right corneal reflex active, but right eyelids did not quite close under ether. Spontaneous spasm of risorius caused

retraction of angle of mouth; spasm spread slightly to upper lip muscles. Faradic Current: Good response in upper lip muscles; less response in lower lip muscles and in orbicularis palpebrarum. No response in risorius. Galvanic Current (6 ma., anodal closure): Equal response on both sides. Stimulation of Rolandic Cortex of both Hemispheres; Left and Right Facial Areas: All muscles responded on both sides, but on stimulation of right facial area, stronger and wider contractions occurred in muscles of left side of face than those observed on right side when left facial area was stimulated.

Brain removed and nerves exposed in posterior fossa. Right Facial Nerve: All muscles contracted. Left Facial Nerve: Muscular contractions stronger on this side. Dissection of Axilla: Ends of Bell's nerve could not be brought together; hence they were grafted on to intercostohumeral nerve in two places. Faradic stimulation of nerve proximal to anastomoses caused contraction of serrations of serratus magnus.

EXPERIMENT 22.—Baboon. Operation on April 9, 1931: Ten millimeters of Bell's nerve substituted for 10 mm. of facial nerve in aqueduct (right side). This monkey is being kept for further observation.

EXPERIMENTS 23 to 28.—Final examinations are included in previous reports. Cat in experiment 28 is being kept for further observation.

EXPERIMENT 29.—Rhesus monkey. Operation on April 1, 1931: One 7 mm. graft of Bell's nerve and two 7 mm. grafts of intercostohumeral nerve substituted for 7 mm. of facial nerve in aqueduct (right side). Final examination on July 17, 1931. No sign of palsy. Winking and corneal reflexes equal on both sides. No spasmodic twitching of muscles. Monkey anesthetized (107 days). Faradic Current: Good response in upper and lower lips. Response in orbicularis palpebrarum did not close right eyelids. Contraction of occipitofrontalis not obtained.

Brain removed and nerve stimulated in posterior fossa. Right and Left Facial Nerves: Equal and full response. Dissection of Axilla: Bell's nerve stimulated proximal to suture. Good contractions of serratus magnus.

EXPERIMENT 30.—Rhesus monkey. Operation on March 30, 1931: A 7 mm. graft of Bell's nerve and two 7 mm. grafts of intercostohumeral nerve substituted for 7 mm. of facial nerve in aqueduct (right side). Final examination on July 21, 1931 (113 days). Face appeared normal. Protective wink on right and left side equal. Right side of upper lips moved equally with left. Monkey anesthetized: Spontaneous twitch of right upper lip muscles. Faradic Current: On right side, good response of upper and lower lip muscles. Partial contraction of right orbicularis palpebrarum and occipitofrontalis as compared with response of these muscles on left side. Left Rolandic Cortex Exposed and Stimulated; Facial Area: Good response in muscles of right side of upper and lower lips, but right eyelids did not close.

Brain removed and nerves stimulated in posterior fossa. Right and Left Facial Nerves: Equal and full contraction of facial muscles.

EXPERIMENT 31.—Rhesus monkey. Operation on March 2, 1931: Great auricular-facial end-to-end anastomosis (right side). Final examination on July 16, 1931 (136 days). Face symmetrical. Slight protective wink on right side. On tickling right pinna no response. Monkey anesthetized. Faradic Current: Definite contraction of orbicularis oris, also of muscles of lower lip. At this moment animal died.

Brain removed and nerves stimulated in posterior fossa. Right Facial Nerve: No response. Left Facial Nerve: Full contraction of all muscles. On further examination it was discovered that there was no facial nerve protruding from right internal auditory meatus; it had been avulsed from side of pons.

EXPERIMENT 32.—Rhesus monkey. Operation on March 4, 1931: Transverse cervical-facial end-to-end anastomosis (right side). Final examination on July 16, 1931 (134 days). Slight droop of right side of upper lip. Slight protective wink on right side. Monkey anesthetized. Faradic Current: Distinct response in all muscles of upper and lower lips. Good response of orbicularis oris at right angle of mouth. No response in orbicularis palpebrarum. No movement of right facial muscles when electrode was placed over stylomastoid foramen. Galvanic Current (6 ma., anodal closure): Larger and sluggish contractions of muscles on right side as compared with left.

Brain removed and nerves stimulated in posterior fossa. Left Facial Nerve: Full contraction of all muscles. Right Facial Nerve: Doubtful slight response in upper lip muscles. Are ends of facial nerve reuniting? Microscopic examination will provide answer.

EXPERIMENT 33.—Rhesus monkey. Operation on March 5, 1931: Great occipital-facial end-to-end anastomosis (right side). Final examination on July 15, 1931 (132 days). Animal winked only with left eye. Slight drooping of right side of upper lip. No movement of face or pinna on stroking or tickling. Faradic Current: Definite response in orbicularis oris; very slight response in muscles of upper lip. Right and Left Rolandic Cortex Exposed and Stimulated; Left Facial Area: No movement of muscles of right side of face. Right Facial Area: Good contraction of muscles of left side of face. Stimulation of Sympathetic Center in Right Hypothalamus: Enlargement of right pupil.

Brain removed and nerves stimulated in posterior fossa. Left Facial Nerve: Full contraction of all facial muscles. Right Facial Nerve: Slight contraction of lip muscles. Severed ends of facial nerve apparently reuniting.

Comment.—It is evident that in experiments 32 and 33 the facial nerve was not effectively removed from the aqueduct. What is still more interesting is that gold leaf was not used in either experiment to block the canal, a piece of muscle being employed in each case to stop the flow of cerebrospinal fluid.

EXPERIMENT 34.—Rhesus monkey. Operation on April 8, 1931: Auriculotemporal-facial end-to-end anastomosis (right side). Final examination on July 20, 1931 (103 days). Protective wink of right eye, associated with elevation of right side of upper lip and movement of right pinna. Protective wink on left side was not associated with any lip or pinna movement. Monkey anesthetized. Faradic Current: Good response all around right side of mouth. Very slight response in orbicularis palpebrarum. Right and Left Rolandic Cortices Exposed and Stimulated. Left Facial Area: No response in muscles of right side of face. Right Facial Area: Good contraction of muscles of left side of face.

Brain removed and nerves stimulated in posterior fossa. Right Facial Nerve: No response. Left Facial Nerve: Good contraction of all muscles. Right Fifth Nerve: No movement in muscles of face or in those of mastication. Left Fifth Nerve: Contraction of left masseter, temporal muscles, etc.

EXPERIMENT 35.—Rhesus monkey. Operation on March 28, 1931: Ten millimeters of trunk of facial nerve outside stylomastoid foramen replaced by 10 mm. of Bell's nerve (right side). Final examination on July 21, 1931 (115 days). Protective wink of both eyes equal, but on right side the upper lip was elevated at same time. There was no lip movement on left side. Face appeared normal. No appearance of palsy. On yawning, lips moved equally, but movement was not quite synchronous. Monkey anesthetized. Faradic Current: Excellent contraction of muscles of upper and lower lips and of orbicularis palpebrarum. Exposure and Stimulation of Right and Left Rolandic Cortices, Left Facial Area: Elevation of right side of upper lip and distinct contraction of orbicularis palpebrarum and of occipitofrontalis. Right Facial Area: Full contraction of muscles of left side of face.

Brain removed and nerves exposed in posterior fossa. Right and Left Facial Nerves: Contractions on both sides of face appeared equal.

EXPERIMENTS 36, 37, 38 and 39.—The final clinical examinations are described in the previous notes.

INFECTIONS OF THE THROAT DUE TO HEMOLYTIC STREPTOCOCCI AND THEIR RELATION TO ARTHRITIS AND ARTHRALGIA

I. OBSERVATIONS WITH PARTICULAR REFERENCE TO STREPTOCOCCUS EPIDEMICUS OF SEPTIC SORE THROAT *

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The etiologic relationship of streptococci of the throat to various forms of arthritis has been the subject of many investigations. That streptococci are the cause of arthritis has the firm support of many clinicians and research workers. The difficulties have been in the efforts of some to establish a specific streptococcus for certain types of rheumatic arthritis. The result has been a lack of uniformity in bacteriology in various forms of arthritis. The problems would not be so confusing if satisfactory differential features could be established for so-called rheumatic fever, acute rheumatic polyarthritis, infectious arthritis, rheumatoid arthritis, arthritis deformans, etc.

Three types of streptococci have been described as responsible, isolated either from the blood or joints, or from the responsible infectious foci. They are the hemolytic streptococcus, Streptococcus viridans and an indifferent, nonhemolytic or nonmethemoglobin-forming streptococcus. It is not my purpose to analyze these streptococci in relation to arthritis. Suffice it to say that there is considerable divergence of opinion among workers as to the relative importance of the various types.[1] Most recent observers have focused their research on the nonhemolytic type. My purpose is to bring forth the importance of hemolytic streptococci from infections of the throat in certain arthritic manifestations and to emphasize the possible rôle of hypersensitiveness to these organisms.

Davis,[2] several years ago, noted the frequent association of hemolytic streptococci in predominant numbers or pure culture in the tonsils of arthritic patients. In recent years, Andrews, Derick and Swift [3]

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* From the Department of Pathology and Bacteriology, University of Illinois, College of Medicine.

1. Jordan, E. P.: The Microbic Etiology of Rheumatic Fever and Arthritis, Arch. Path. **10**:79 (July) 1930.

2. Davis, D. J.: Bacteriology and Pathology of the Tonsils with Especial Reference to Articular, Renal and Cardiac Lesions, J. Infect. Dis. **10**:148, 1912.

3. Andrews, C. H.; Derick, C. L., and Swift, H. F.: Streptococci in Acute Rheumatic Fever with an Analysis of the Antigenic Relationships Existing Among Certain Strains, J. Exper. Med. **43**:13, 1926.

also pointed out the importance of hemolytic streptococci, but found no homogeneity among the twenty-seven strains isolated from their patients, when studied by agglutination and absorption tests and by protection experiments.

At the present time three clinical conditions seem to be due to distinct types of hemolytic streptococci, namely, scarlet fever, erysipelas and septic sore throat. Streptococcus scarlatinae, Streptococcus erysipelatis and Streptococcus epidemicus of septic sore throat can be fairly well differentiated either culturally and immunologically, particularly by agglutination, the opsonic method, or by skin toxin and neutralization tests in susceptible persons. In all of these clinical entities arthritis as a complication or sequela may occur. In scarlet fever, attacks of rheumatism identical with acute rheumatic fever usually occur at the end of the third or during the fourth week. In some instances nonseptic arthritic symptoms develop earlier on from the fifth to the seventh day, when the rash is fading. In erysipelas, arthritic pains may be observed early, or more often in later stages of the disease. In epidemic septic sore throat, Davis [4] noted arthritis as a complication and as a later sequela. Another interesting observation made on the relationship of hemolytic streptococci to the arthritic pains was well illustrated in two patients I observed with severe streptococcal infections of the hand. During the third week or later, when the hemolytic streptococci were still demonstrable in the wound, arthritic pains of a wandering type appeared. With gradual improvement and complete healing, the pains disappeared.

It is evident that the arthritic manifestations may develop after infections by various hemolytic streptococci and do not depend on a specific streptococcus. Indeed, if one looks further, one finds that in practically all cases first attacks of acute rheumatic fever are preceded by either sore throat or infection of the upper respiratory tract. Recurrent types of arthritis are often associated with recurrent sore throat or "cold." In these conditions the arthritic manifestations vary from the more typical acute rheumatic fever to very mild migratory pains in the muscles and joints without gross signs of inflammation.

In the course of investigations of sporadic cases of septic sore throat due to Streptococcus epidemicus, I noted acute arthritis in three instances during convalescence. Arthralgia was observed in several cases, appearing after apparent recovery from acute sore throat and persisting for several months (table 1). From bacteriologic studies the arthritic manifestations appear to be due to Streptococcus epidemicus in the tonsils or throat. During the acute stage, this streptococcus was obtained in predominant or pure numbers. In three patients with acute arthritis, the streptococci were found in pure culture

4. Davis, D. J.: Septic Sore Throat, Oxford Medicine, Philadelphia, W. B. Saunders Company, 1920, vol. 2, p. 1.

in the throats, two of which were devoid of tonsils. The arthritis disappeared simultaneously with the disappearance of Streptococcus epidemicus from the throat. In the patients with persistent arthritic pains, the streptococci were found in the tonsils usually in predominant numbers, and with the removal of the tonsils, the arthritic pains disappeared. Later Streptococcus epidemicus could not be demonstrated in the tonsillectomized throat.

In this connection it would be well to point out that investigations on S. epidemicus in relation to arthritis are more significant if certain data are presented about this organism. In the first place, hemolytic streptococci occur in the throats of 61 per cent of persons [5] and almost 97 per cent of excised tonsils. This common occurrence has been the basis for doubting the etiologic relationship of these streptococci to arthritis. On the other hand, S. epidemicus in routine cultures from throats was found in less than 1 per cent [6] (4 of 500 persons), and 3 of the 4 carriers of these streptococci had arthritic symptoms. One may assume that when this organism is found in the throat there may be an etiologic connection to the associated pathology. The ease with which throat cultures can be followed by the use of ascitic blood agar,[7] which readily differentiates S. epidemicus from other hemolytic streptococci, adds another valuable feature to the study of the appearance and disappearance of these organisms from the throat in connection with the onset, course and termination of the arthritic symptoms. This streptococcus forms large, moist or flat colonies which in wet preparations with india ink reveal encapsulated cocci.

The acute cases of polyarthritis associated with a positive blood culture of S. epidemicus are of great interest.

REPORT OF CASES

Case 1.—In Sh., a boy, aged 10 (Edgewater Hospital), who was convalescing from scarlet fever, a chill developed on the twenty-fifth day; the temperature was 105 F.; on the second and sixth days, by ordinary methods, hemolytic streptococci were cultivated from the blood. The streptococci formed colonies like S. epidemicus. Polyarticular involvement of the inflammatory and nonsuppurative type then developed. The tonsils were large and red and yielded similar streptococci. The fever and arthritis subsided after four weeks, but two weeks later recrudescence of the fever (102 F.) and a milder arthritic involvement occurred. Autogenous vaccines gave marked skin reactions. This patient recovered after twelve weeks.

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5. Pilot, I., and Davis, D. J.: Hemolytic Streptococci in the Faucial Tonsil and Their Significance as Secondary Invaders, J. Inct. Dis. **24**:389 (April) 1919.

6. Pilot, I.; Hallman, B., and Davis, D. J.: Human Carriers of Streptococcus Epidemicus, J. Infect. Dis. **47**:503 (Dec.) 1930.

7. Pilot, I.; Hallman, B., and Davis, D. J.: A Culture Medium for the Isolation of Streptococcus Epidemicus of Septic Sore Throat, J. A. M. A. **95**:264 (July 26) 1930.

TABLE 1.—*Sore Throat Due to Streptococcus Epidemicus Complicated by Arthritis* *

Name	Age	Sex	Previous History	Clinical Symptoms	Course	Result
A. C.	30	M	Sore throat 3 weeks previously	Arthritic pains; large edematous tonsils	Excised tonsils contained many S. epidemicus	No report
L.	32	F	Attack of rheumatism 3 years previously	Red pharynx (tonsils out); acute polyarthritis; pure culture of S. epidemicus from throat	Twelve days	Complete relief; subsequent throat cultures negative for S. epidemicus
R.	10	M	Repeated sore throats	Red pharynx (tonsils out); acute polyarthritis; complete relief; almost pure culture of S. epidemicus	Ten days	Streptococci disappeared completely with relief of arthritis
F.	20	M	Follicular tonsillitis; S. epidemicus in predominant numbers	Arthritic pains	Complete relief after tonsillectomy; S. epidemicus in excised tonsils	Subsequent throat cultures negative
Mc.	50	M	Repeated polyarthritis	Acute tonsillitis; acute polyarthritis; S. epidemicus in throat culture	Improved after tonsillectomy; excised tonsils had S. epidemicus	Cultures negative; markedly improved
S.	30	M	Sore throat previously; pure S. epidemicus	Arthritic pains; enlarged red tonsils	Tonsillectomy; cultures positive in tonsils	Relief after tonsillectomy; cultures negative
E. P.	19	F	Tonsillitis due to S. epidemicus	Arthritic pains	Tonsillectomy; cultures positive in tonsils	No pains; cultures negative
W. D.	17	M	Repeated attacks of rheumatism and sore throat	Red pharynx; acute polyarthritis; (tonsils out); pure culture of S. epidemicus at onset	Recovery 14 days	Throat cultures negative
C. H.	..	..	Upper respiratory infection	Acute polyarthritis (tonsils out); blood culture positive for S. epidemicus	Three weeks' duration	Apparent complete recovery; cultures negative
Sh.	..	..	Scarlet fever; onset 25 days previously	Acute polyarthritis; blood culture positive for S. epidemicus	Throat culture persistently positive	Recurrence with fever of 102 F. after being markedly improved for 2 weeks

* From Cook County, Lutheran Deaconess and Edgewater hospitals.

CASE 2.—Se., a colored man, aged 30 (Cook County Hospital), had an infection of the upper respiratory tract; chill, fever and polyarthritis developed. He had had rheumatism three years previously. A blood culture by ordinary methods yielded S. epidemicus. The pharynx was red and devoid of tonsils. The fever and arthritis subsided, and the patient appeared to be making a complete recovery.

Most interesting was the occurrence of acute polyarthritis in two other patients who previously had had rheumatism.

CASES 3 and 4.—In L. and D. (Cook County Hospital), both of whom had a previous history of rheumatism, sore throat and polyarthritis of the rheumatic type developed; the temperature did not exceed 102 F. In from ten to fourteen days, the condition subsided completely. Both patients had enlarged hearts indicative of an old endocarditis. The tonsils were absent. The throats were red, and in both cases almost pure cultures of S. epidemicus were obtained; with recovery, the throat cultures became negative.

These cases demonstrate the importance of the underlying susceptibility or hypersensitiveness of the patient to streptococci. The earlier attacks may have been due to other streptococci, but when a fresh infection of the throat occurred, the tendency to arthritic complication again manifested itself.

The complete relief in three cases, in contrast to the first case, was due to the disappearance of the organism from the throat. In the one case the streptococci persisted in the tonsils and the arthritis was persistent for four weeks and then recurred in milder form. In the others, in which the throats were devoid of tonsils, the streptococci promptly disappeared, and the lack of establishment of a focus may account for the complete recovery from the arthritis. This is further illustrated in the patients who had persistent joint pains after sore throat due to S. epidemicus. In these patients (table 1), cultures from the exudate from the tonsils during the acute sore throat yielded S. epidemicus in predominant numbers or in pure culture. On recovery, it appears that in a certain number of patients the organism persisted in the tonsils and the person became a carrier. As pointed out in previous work, many of the carriers have arthritic pains. The simplest treatment is to remove the tonsils, which terminates the carrier state.[8] As a result of the removal of the tonsils, S. epidemicus disappears, and the patient often becomes completely relieved of the arthritic symptoms.

In previous work,[6] 506 pairs of tonsils were examined for S. epidemicus, and the organism was found in 70 (13.8 per cent). In 23 of the 70, arthritis or arthritic pains were cited as the cause for removal in addition to a history of sore throats. Nineteen of these patients are listed in table 2. From an analysis of this table it was

8. Pilot, I., and Davis, D. J.: Tonsillectomy in Carriers of Streptococcus Epidemicus, J. Infect. Dis. **48**:501 (May) 1931.

TABLE 2.—*Patients with Arthritis and Arthralgia Whose Excised Tonsils Contained Streptococcus Epidemicus* *

Name	Age	Sex	Date of Tonsillectomy	Clinical History	Results and Date of Last Report	Comment
A. L.	28	F	8/12/30	Rheumatic pains	Relieved completely 3/14/31	
W. E.	9	M	8/12/30	Sore throat; rheumatic pains	Complete relief 3/14/31	
B.	20	F	5/20/30	Sore throat; pains in joints	Complete relief 4/11/31	
A.	21	F	6/30/30	Sore throat; pains in joints	Complete relief 3/14/31; slight recurrences	
L. K.	4	F	7/ 3/30	Sore throat; joint pains	Complete relief 10/20/30	
M. F.	19	F	7/17/30	Muscle and joint pains	Marked improvement 9/12/30	
M. G.	20	M	7/16/30	Sore throat; muscle pains	Complete relief 12/10/30	
A. G.	3	M	7/31/30	Cervical adenitis; general pains	Relieved 12/10/30; adenitis gone	
R. M.	7	M	8/16/30	Sore throat and joint pains	Relieved 12/10/30	Excised tonsils revealed pure cultures of S. epidemicus
R. I.	48	M	8/18/30	Sore throat; rheumatic pains	Complete relief 4/14/31	
E. H.	17	F	8/18/30	Rheumatic pains	Relieved 4/14/31	Excised tonsils revealed pure cultures of S. epidemicus
E. M.	6	F	8/21/30	Muscular pains; otitis media	Complete relief 4/14/31	
V. K.	26	F	9/26/30	Rheumatic pains	Relieved 11/10/30	
E. P.	19	F	5/14/30	Cervical adenitis; rheumatic pains	Relieved 6/14/30	4/14/31, pains returned after cold and sore throat; cultures; no epidemicus; few hemolytic streptococci; many Strep. viridans
J. K.	50	M	8/16/30	Chronic polyarthritis of inflammatory type	Partial improvement 10/7/30	
E. L.	50	F	8/18/30	Sore throats; arthritis of inflammatory type	Flareup 2 weeks after tonsillectomy; later complete relief, 9/15/30	
C. K.	26	F	7/30/30	Sore throats; polyarthritis of inflammatory type	Flareup 2 weeks after tonsillectomy; no subsequent report	
C. P.	25	F	7/15/30	Quinsy, arthritis of inflammatory type	Complete relief 3/17/31	
M. T.	57	F	7/15/30	Marked deforming arthritis; bedridden	Up and about; marked improvement 11/28/30	
L. N.	34	F	7/31/30	Tonsillitis and arthritis	Improved 9/18/30	
A. D.	28	M	9/ 3/30	Inflammatory arthritis and endocarditis	Flareup after tonsillectomy; marked improvement 3/19/31	

* From Lutheran Deaconess and Edgewater hospitals.

observed that tonsillectomy gave complete relief to those with arthritic pains and relief or improvement to those with inflammatory joints. Two types of arthritis were evident: one inflammatory of several days or weeks' duration, and the other apparently noninflammatory by gross examination and characterized by fleeting migratory pains. The former may well be due to the stretococci deposited in the joints in small numbers, but the latter can hardly be attributed to infecting organisms, as the joint manifestations are only of a few hours' duration. Bacterial products or toxins may perhaps be the cause of such vagrant pains. This hypothesis has some basis in the experience of workers on immunization with the toxin of streptococcus of scarlet fever. Arthritic pains are not uncommon the day following an injection of toxin. Of 45 persons I have immunized, arthritic pains appeared in 1 after the second injection; in 5 after the third, and in 4 after the fourth. In all no previous rheumatic pains had been noted. The pains were polyarticular and muscular and disappeared within twenty-four or forty-eight hours.

With the acceptance of the streptococcus as the common cause of infectious arthritis, investigators have directed their attention to the mechanism of the infection. Several peculiarities in the development appear to stand out. In the first place, arthritis develops in certain persons in the course of recovery from other diseases at an interval of from one to four weeks from the onset of the primary disease; secondly, arthritis has a cyclic character, with periods of exacerbation and abatement of symptoms. Hypersensitiveness or allergic reactions to the streptococcus have been suggested; for this hypothesis, some experimental data with reference to hemolytic streptococci are available. Using the streptococcus of scarlet fever, Zinssner [9] gave guinea-pigs injections of living streptococci, filtrates and streptococcal nucleoproteins. Intracutaneous allergic reactions were obtained about three weeks later. The best results were observed from sensitizing injections of living streptococci intraperitoneally and testing with filtrates intradermally.

Dochez and Stevens [10] gave rabbits injections of filtrates of Streptococcus erysipelatis; they described two types of hypersensitiveness in animals, one to a toxin that can be neutralized in vitro, and a second to a bacterial thermostabile product that they were unable to neutralize.

My associates and I are in the midst of some experiments dealing with this question. During a course of intravenous injections of killed suspensions of Streptococcus epidemicus for the production of agglu-

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9. Zinsser, H., and Yu, H.: The Bacteriology of Rheumatic Fever and the Allergic Hypothesis, Arch. Int. Med. **42**:301 (Aug.) 1928.

10. Dochez, A. R., and Stevens, F. A.: Allergic Reactions with Strains from Erysipelas, J. Exper. Med. **46**:487, 1927.

tinins, the rabbits have revealed no arthritic manifestation; similarly, rabbits given injections of broth filtrates of the streptococcus for the production of antitoxic serum have not reacted. A rabbit that had received the dead streptococci was then given injections of minute doses of living streptococci intravenously at weekly intervals. After the third injection, a swelling of the front elbow joint developed. A new series of small rabbits was then given intravenous injections of variable doses of living streptococci. The first dose of from 0.1 or 0.2 to 0.3 cc. did not cause arthritis. Another series of four rabbits was given 0.01 cc. of living streptococci at weekly intervals. After the third dose, one rabbit developed a swelling of a front wrist joint from which, on aspiration, the purulent material on culture contained the streptococci in sparse numbers. Another developed a joint swelling after the fourth injection. Four rabbits previously given injections of vaccine or filtrates received a similar single small dose of living streptococci, but arthritis did not develop. It appears that this sensitization to arthritis can be produced by the living streptococci and apparently not by the killed cocci or filtrates, when injected intravenously. In the skin reactions on these rabbits, those given injections of the living streptococci gave indurated lesions to the vaccine injected intradermally but little or no reaction to filtrates.

Intradermal tests have been performed in large numbers with filtrates of nonhemolytic streptococci. Kaiser [11] found that only 15 per cent of normal children react positively, but that 70 per cent of rheumatic children give positive reactions. With the extracts of hemolytic streptococci, MacKenzie and Hanger [12] obtained good reactions in many arthritic patients. These were not successfully produced by passive transfer, or neutralized by rabbit immune serum. My own observations with skin reactions in rheumatic patients are incomplete. I was unable to obtain constant reactions in the arthritic patient with the dilute toxin of hemolytic streptococci. The undiluted toxin and vaccine produced a considerable number of reactions in both normal and rheumatic persons. The latter patients gave most constant and often strong reactions to the vaccines.

In cases of arthritis characterized by inflammatory lesions, it is possible that the patient becomes sensitized by the living streptococcus from a focus like the tonsil and renders him susceptible to minute numbers of streptococcus which produce violent reactions. In arthralgia perhaps a similar hypersensitiveness develops from the toxins from a

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11. Kaiser, A. D.: Skin Reactions in Rheumatic Fever, J. Infect. Dis. **42**:25 (Jan.) 1928.

12. MacKenzie, G. M., and Hanger, F. M.: Allergic Reactions to Streptococcus Antigens, J. Immunol. **13**:41, 1927.

focus containing the living organisms. In either case, if the focus, like the tonsils harboring the responsible streptococcus, is removed and the streptococcus disappears from the throat, improvement or cure will be expected and often is obtained. Such a focus need not show abscesses or thrombosed vessels indicative of infection under tension or with inadequate drainage. Histologic examinations of the tonsils with S. epidemicus from arthritic patients revealed signs of chronic inflammation, often with cocci in the crypts, but seldom demonstrable under the epithelium. The changes in these tonsils, however, were no more marked or different from tonsils harboring the same streptococcus in patients without arthritis. These findings indicate that while a focus is necessary the important underlying factor for the development of arthritis may be the hypersensitive or allergic state of the patient.

CONCLUSIONS

Arthritis and arthralgia may complicate scarlet fever, erysipelas, wound infections and sore throat due to hemolytic streptococci.

That the complicating arthritis develops three or more weeks after the onset of the streptococcal disease suggests hypersensitiveness as an important factor.

Streptococcus epidemicus of septic sore throat may be responsible for a relatively small number of cases of acute and chronic arthritis of the so-called rheumatic type.

The persistence of Streptococcus epidemicus in the tonsils is often associated with arthritis. Removal of the tonsils terminates the carrier state, and the associated arthritis frequently disappears.

THE NATURAL ORIFICE OF THE MAXILLARY SINUS

I. ANATOMIC STUDIES *

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This paper is based on a study of 114 lateral nasal walls of adult heads in the fresh state. The position, size, shape and relations of the maxillary ostium were noted. Special attention was paid to its accessibility for sounding and for irrigation. The presence of accessory ostia was sought for and noted.

The situation of the ostium in the posterior portion of the infundibulum ethmoidale where the bulla tapers away on the upper side and the uncinate on the lower side makes it desirable to know these structures, all of which are located under cover of the middle turbinate.

THE UNCINATE PROCESS

The uncinate process of the ethmoid bone is a fine, thin, delicate bony process originating from the anterior medial aspect of the ethmoid labyrinth. It is covered by the middle turbinate and is in relation to the medial and posterior surface of the lacrimal bone. Its length is given by Schaeffer [1] as from 15 to 20 cm. In this study the shortest was 14 mm. and the longest 22 mm. This structure curves downward, backward and laterally, its crescentic curve being parallel to that of the bulla. The uncinate process constitutes the convex downward side of the hiatus semilunaris. It covers part of the hiatus maxillaris, and it articulates with one or more ethmoidal processes of the inferior turbinate bone. The bony uncinate process turns upward behind the posterior end of the infundibulum, frequently causing a slight depression. When covered with mucous membrane, this upward end is not prominent. The lower posterior end is usually quite prominent, however.

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1. Schaeffer, J. P.: The Nose, Paranasal Sinuses, Naso-Lacrimal Passageways and Olfactory Organ in Man, Philadelphia, P. Blakiston's Son & Co., 1919, chap. 3.

The uncinate is the medial boundary of the hiatus semilunaris. Its width or height denotes the depth of the infundibulum. It might be called the wall or fence that guards the infundibulum. According to my observation, the uncinate was absent three times; rudimentary, ten times; moderately prominent, sixty-nine times, and pronounced, thirty-two times. The term "pronounced" was used for those greater than 5 mm. in height. It was noted that the uncinate process was usually extremely thin and nonresistant. In 5 specimens it was found abnormally thick and resistant. A great majority of the uncinate processes seen in this study were so delicate and fragile, because of the extreme thinness of their bony structure, that they offered no resistance to displacement by an instrument.

THE BULLA ETHMOIDALIS

The bulla ethmoidalis is a convex bony structure that may be considered to be the floor of the anterior ethmoidal labyrinth. It forms the most medially placed cell boundary, and is the most prominent structure in the middle meatus. It is usually situated in its anterior half. It lies hidden by the anterior portion of the middle turbinate and can be seen only when this structure is removed or turned upward out of the way. Occasionally, the middle turbinate does not reach down far enough to cover this structure so that it may then be seen. It may also be seen when it is abnormally prominent. This is exceptional. It acts as the medial wall for one or more ethmoidal cells. It forms the lateral and superolateral boundary of the hiatus semilunaris, and its proximity to the uncinate process governs the width of this space. Thus the bulla may touch (seldom) the uncinate, or it may be separated by several millimeters from it. In this study there was not a single specimen in which the bulla touched the uncinate, although in one the space between these structures was less than a millimeter. The greatest distance noted between the bulla and the uncinate was 3 mm. The bulla has a crescentic curve that is parallel with that of the uncinate except posteriorly. Here the posterior curve of the bulla usually takes an upward turn, which widens the space between it and the uncinate, which takes a downward curve.

THE HIATUS SEMILUNARIS

The hiatus semilunaris is the space between the bulla and the upper border or free edge of the uncinate process. It can be considered as a cleft opening into and following the course of the infundibulum. In this study the structure varied from 14 to 22 mm. in length and from 0.5 to 3 mm. in width. The hiatus semilunaris ends where the uncinate diverges from the bulla. The posterior extremity of the hiatus is in direct continuity with the membranous portion of the middle meatus.

THE INFUNDIBULUM ETHMOIDALE

The infundibulum, also called the uncinate groove by Mosher,[2] is the trough, depth, furrow, groove or channel caused by the proximity of the bulla ethmoidalis to the uncinate process. It constitutes a crescentic depression, the shorter boundary of which is made by the bulla, while the longer boundary is made by the uncinate. It is the bottom of the cleft that the hiatus semilunaris represents. It begins anteriorly and above as a blind depression, which is usually an anterior ethmoidal cell, or it may be the continuation of the nasofrontal passage. Its posterior and lower end where the ostium is found loses its identity by gradually becoming more shallow and flattening out until it merges into the surface of the middle meatus. Occasionally a small rounded depression is encountered directly posterior to the termination of the infundibulum. This is formed by the upper posterior terminal of the uncinate when such a process exists. The infundibulum becomes wider as it extends posteriorly. The depth of the infundibulum depends on the height of the uncinate process. This varied from 0.5 to 10 mm. in this study.

The angle formed by the distal upward curve of the bulla and the distal downward curve of the uncinate is present in almost all specimens. In some it is placed more forward than others, depending on the distance between the uncinate and the bulla before they separate posteriorly. This bulla-uncinate angle is the anatomic guide to the maxillary ostium.

THE MIDDLE TURBINATE OR CONCHA MEDIA

The middle turbinate overhangs the foregoing structures. The lower and posterior end of the hiatus semilunaris approximates the free border of the middle turbinate very frequently. The upper end of the hiatus semilunaris is well covered by this structure and is usually out of sight. Occasionally the middle turbinate does not extend far downward enough to cover the uncinate, so that at times not only the uncinate but also the hiatus and bulla may be seen. The distance of the ostium from the anterior free end of the middle turbinate was noted in each lateral wall.

The anterior edge of the ostium in relation to the anterior border of the middle turbinate was found to be as follows in 111 specimens: 2 mm. in front, 2 specimens; even with, 6; from 1 to 10 mm. behind, 48; from 11 to 20 mm. behind, 53; 22 mm. behind, 1, and 23 mm. behind, 1.

PARS MEMBRANACEA

The membranous portion of the lateral nasal wall is situated posteriorly to the posterior end of the hiatus semilunaris and represents the

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2. Mosher, H. P.: The Surgical Anatomy of the Ethmoidal Labyrinth, Tr. Am. Acad. Ophth., 1929.

unossified area between the bony parts of the lateral wall of the nose. Dr. Senior believes it is better to regard the pars membranacea as the region of the primitive nasal cavity into which ossification has spread.

This space is bounded anteriorly by the lacrimal bone, and posteriorly by the palate bone, above by the horizontal plate of the maxilla and below by the upper end of the inferior turbinate. Anteriorly, it is also bounded by the posterior end of the uncinate process. The membranous portion also fills the space between the posterior end of the uncinate and the bulla. In it there is a small opening, the maxillary ostium. The membranous wall in the remainder of the space is covered by two thicknesses of mucous membrane in apposition with each other, constituting a nasal and a maxillary surface.

LAMINA PAPYRACEA

The lamina papyracea is also called the os planum or orbital plate of the ethmoid. This most lateral boundary of the ethmoid bone articulates with the perpendicular plate of the frontal bone above, the lacrimal bone in front and the sphenoid behind. Below, it makes an obtuse angle where it joins the orbital plate of the maxilla. It is just below this obtuse angle that the maxillary ostium is to be found in the extreme upper part of the nasal wall of the antrum. This structure was studied both to observe its thickness and to define its relationship to the structures of the middle meatus. A Pierce cannula was used to demonstrate the resistance to perforation of this structure. Its thickness varied slightly in different skulls. In none of the specimens studied was this structure capable of resisting penetration by a blunt-ended instrument. In a great majority of cases the very fine thinness was apparent. It was demonstrated that an instrument piercing the infundibulum in any location anterior to the maxillary ostium would invariably pass through the lamina papyracea into the orbit (fig. 1).

The following interesting experience was noted with regard to the vulnerability of this structure. In one lateral wall the passage to the antrum began as a horizontal orifice in the infundibulum, continued as a tube, and ended as a vertical opening in the antrum (fig. 2 *a*). The lamina papyracea formed part of the outer wall of this tube. The Yankauer and Pierce instruments could not be introduced under direct vision with the middle turbinate removed, without piercing this structure and entering the orbit. This is exceptional, for the lateral wall of these tubular passageways is usually the medial surface of the bulla or of the orbital plate of the maxilla.

MAXILLARY OSTIUM

The ostium of the maxillary sinus is located immediately below the orbital floor, and therefore below the lamina papyracea of the ethmoid, in the posterior portion of the infundibulum. It is usually situated just

below the point where the orbital plate of the ethmoid and the orbital plate of the maxilla meet on their medial or internal aspects (fig. 1).

It is all that is left of the bony hiatus maxillaris after the bony structures are articulated and covered by mucosa. It is either buried

Fig. 1.—Diagrammatic drawing showing the relation of the infundibulum and the maxillary ostium to the lamina papyracea, orbital plate and surrounding structures.

Fig. 2.—Semidiagrammatic drawing showing variety of nasomaxillary passageways.

deeply or can be readily approached, depending on its relation to the infundibulum and the accessibility of this structure. This relationship of the ostium to the uncinate and infundibulum determines its availability for irrigation of the sinus. When the ostium is deeply placed, it

is rarely hidden by the bulla. When it is inaccessible, it is invariably made so by the high obstructing wall which the uncinate represents. In 14 of the 114 specimens, the ostium was so hidden by the uncinate as to preclude the possibility of passing a properly bent probe into it from the anterior nasal orifice. In these instances the height of the uncinate wall was usually 5 mm. or more. In one its height was 10 mm. In another specimen, where the anterior structures were absent, the

Fig. 3.—Variety of maxillary ostia: *A*, ostium extending above the level of the free border of the uncinate; *B*, ostium entirely independent of the uncinate; *C*, double maxillary ostium; *D*, wide maxillary ostium replacing the uncinate and bulla, and *E*, ostium occupying the entire infundibulum.

ostium was hidden by a curtain of mucosa so that 15 or 13 per cent of the ostia were inaccessible. In 6 specimens the natural orifice was found entirely free and independent of the infundibulum, so that the height of the uncinate and the depth of the infundibulum did not matter (fig. 3 *B*). In 5 specimens the ostium occupied the entire infundibulum (fig. 3 *D* and *E*). In 2 specimens the ostium occupied the center of the infundibulum.

The maxillary ostium lies either in the depth of the infundibulum or is to be found in its lateral wall. When lying in the depth of the infundibulum, the ostium is horizontally or obliquely placed. When found in the lateral wall, it is vertically placed. In this study the maxillary ostium was horizontally placed, fifty-five times; vertically placed, fifty-one times, and obliquely placed, eight times.

Gosselin [3] is the only writer on the subject of the maxillary ostium who mentions its horizontal and vertical placement in these terms. The ostia that are vertically placed are usually easily accessible. The horizontally placed ostia are more difficult to enter because of their deeper position. It is the horizontal and oblique ostia that represent the nasal beginning of a passageway into the antrum. These passageways extend

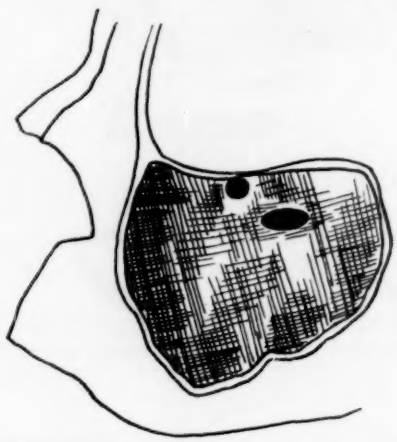

Fig. 4.—Nasal wall of the antrum showing usual location of the ostium. An accessory ostium is present.

between the bulla and the uncinate or between the orbital plate of the ethmoid and the uncinate, or between the orbital plate of the maxilla and the uncinate. They usually terminate in a vertical orifice in the wall of the maxillary sinus (fig. 4).

In many instances the ostium that is horizontally placed in the infundibulum assumes a different form on the maxillary sinus wall. In such instances the approach to the antral cavity from the nasal orifice is by means of a tubular passage, at times S-shaped, at times funnel-shaped, almost straight, obliquely vertical, obliquely horizontal or horizontal. Because there is a definite tubular or funnel-shaped passageway frequently occurring between the middle meatus and the medial

3. Gosselin: Sur l'orifice du sinus maxillaire, Compt. rend. Soc. de biol. **3**:53, 1851.

wall of the antrum, it might be well to speak of this as the nasomaxillary passageway or canal, rather than the maxillary ostium. Because of this tubular type of approach to the antrum, the nasal and antral orifices of the same passageway show differences in size, shape and direction (fig. 2).

The most constant guide to the location of the maxillary ostium is the bulla uncinate angle previously described.

The distance of the maxillary ostium from the floor of the nose and from the roof of the nose was measured in each specimen. The figures show such a wide variation that this measurement is not considered of value. The accessibility of the ostium in each specimen was tested with the Yankauer and Pierce instruments. In 15 instances the ostium was considered inaccessible from the anterior nasal orifice.

Size.—The size of the maxillary ostium varies in the reports of different authors. In a study of 300 bodies, Zuckerkandl [4] found the largest to be 19 by 5 mm. and the smallest 3 mm. in diameter. Oppikofer [5] found the smallest to be 2 mm. in diameter and the largest 17 by 11 mm. in 200 sections. Schaeffer noted measurements of 1 to 22 mm. in length, and 1 to 6 mm. in width in 122 specimens. In this study of 114 specimens the smallest was 1 mm. in diameter, and the largest was 13 by 11 mm. Zuckerkandl called attention to the fact that a large opening to the antrum may exist as a result of the merging of the ostium with an accessory ostium behind it. This is readily understood when one considers that the accessory ostium is separated from the natural ostium by a bridge of membranous tissue. Zuckerkandl, Onodi,[6] Hartmann,[7] Killian,[8] Schaeffer and others have illustrated large ostia that occupy the entire infundibulum.

The following observations are recorded:

1. The vertical placement of the ostium so that its upper portion is found to a greater or lesser extent above the uncinate process insures an easy passage of the cannula in the antrum. In 26 specimens, the ostium was found extending above the edge of the uncinate in this manner (fig. 3 *A*).

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4. Zuckerkandl, E.: Normale und pathologische Anatomie der Nasenhöhle, Leipzig, Wilhelm Braumüller, 1893.

5. Oppikofer, E.: Beiträge zur normale und pathologische Anatomie der Nase und ihrer Nebenhöhlen, Berlin, 1907, p. 28.

6. Onodi, A.: The Anatomy of the Nasal Cavity and the Accessory Sinuses, ed. 2, trans. from ed. 2 by Sir St. Clair Thomson, London, H. K. Lewis, 1895.

7. Hartmann, A.: Atlas der normalen und pathologischen Anatomie der Nase, Berlin, H. Kronfeld, 1891.

8. Killian, G.: Die Krankheiten der Kieferhöhle, Handb. d. Laryng. Rhin. 3:50, 1898.

2. In 6 specimens, the ostium was found lying entirely independent of the bulla and uncinate in a vertical position. In these specimens the cannula could be passed into the antrum readily (fig. 3 *B*).

3. In 5 specimens, the ostium occupied the entire infundibulum (fig. 3 *D* and *E*). In one of these (no. 70) the bulla, uncinate and infundibulum were absent, and this area was replaced by a large vertical opening the anterior boundary of which was the posterior border of the lacrimal bone (fig. 3 *D*).

4. In 2 specimens (82 and 91), the ostium occupied the center of the infundibulum.

5. In specimens no. 7 and 8 there was fusion of the agger nasi, uncinate and bulla with a high anteriorly placed small ostium directly behind the posterior border of the lacrimal bone.

In specimen no. 82 (fig. 3 *C*), a double ostium was found; the more anteriorly placed one was found located in the center of the infundibulum, the second one lying 2 mm. behind it. These ostia led into a single antral cavity.

Heavy copper wire was bent with suitable pliers so as to demonstrate the most direct approach to each ostium from the anterior nasal orifice. The copper wire study revealed that the most suitable probe or sound was one the distal end of which was bent at an angle of 90 degrees, the length of the distal arm being 6 mm.

THE ACCESSORY OSTIUM

The accessory ostium is recorded by various authors as occurring in from 10 to 50 per cent of lateral nasal walls. This ostium leads into the maxillary sinus and represents an opening in the pars membranacea. The frequency of an accessory ostium as found by various authors is shown in table 1.

In this study of 114 specimens there were 35 accessory ostia, or 30.7 per cent. In one of these there were 2 accessory ostia.

The accessory ostium was first mentioned by Giraldès who thought its presence pathologic. Zuckerkandl and others do not agree with Giraldès as to the origin of these ostia; they cite the absence of evidence of a pathologic process in the maxillary sinuses that were found to have an accessory ostium.

If they occur normally, they must be due to development. It would therefore follow that as the true adult form is reached the thinning of the membranous portion of the middle meatus proceeds beyond a point where the lateral wall can remain intact (Schaeffer). This hypothesis is borne out by the fact that accessory ostia are rarely seen in children and infants. One should only expect to find an accessory ostium where

the growth of the maxillary sinus has progressed sufficiently to cause a thinning out of the membranous portion of the nasal wall. This should be after the thirteenth year. Schaeffer and others have not found an accessory ostium in the fetus or young child. Davis found an accessory ostium in a child of 4, and Oppikofer found one in a child of 7, and

TABLE 1.—*Frequency of the Presence of an Accessory Ostium*

Author	Number of Specimens Studied	Number Found	Percentage
Giraldès: Des maladies du sinus maxillaire, Thesis Paris, 1851	100 heads	Not given	8 to 10
Zuckerkandl [4]	300 bodies	Not given	10 to 11
Chiari, quoted by Schaeffer [1]	.....	.....	10
Hajek: Nasal Accessary Sinuses, St. Louis, C. V. Mosby Co., 1929, vol. 1, p. 208	.....	.....	20
Davis: Development and Anatomy of the Nasal Accessory Sinuses in Man, Philadelphia, W. B. Saunders Co., 1914, p. 112	114	.....	15
Anatomical Society Great Britain	152	.....	17.5
Turner: The Accessory Sinuses of the Nose, New York, Longmans Green & Co., 1902	9	4	44.4
Schaeffer [1]	80	35	43.75
Ibid.	125	..	42.4
Ibid.	90	..	37
Oppikofer [5]	400	45	11.2
Hansberg: Monatschr. f. Orenh. 24 : 3, 1890	80 bodies	1 in 6 or 7	14 to 15
Douglass: Nose and Throat Surgery, Philadelphia, F. A. Davis Co., 1906, p. 125	Not stated	.....	50

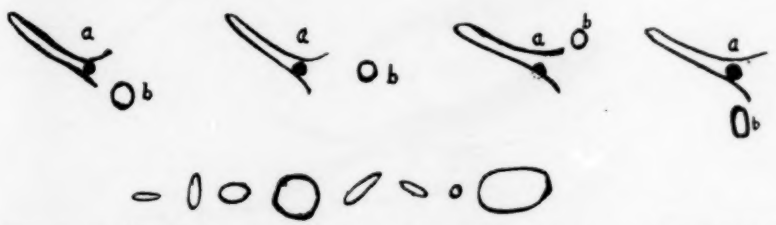

Fig. 5.—Various shapes and positions of the accessory ostium in relation to the normal ostium: *a*, most frequent type, below and behind, eighteen times; *b*, same level behind, eight times; *c*, above and behind, four times and *d*, directly below, five times.

another in one of 11 years of age. Schaeffer also records such an orifice in an 11 year old girl. The accessory ostium is usually single but may be double. Rarely are there three.

Location and Size.—The accessory ostium was found to be located a distance of 1 to 11 mm. from the maxillary ostium in the membranous middle meatus. The positions were as follows: diagonally behind and below, 18; directly behind, 8; directly below, 5, and superior to the maxillary ostium, 4.

The size of the accessory ostium in this study varied from a pinhead to 10.5 by 6.5 mm. (table 2). In two specimens a large accessory ostium reached to the roof of the antrum. In another specimen a large ostium merged with the posterior wall of the antrum.

TABLE 2.—*Size of Accessory Ostia* *

Smallest	Pinhead
Largest	10.5 × 6.5 mm.
Height	1 to 9 mm.
Length	1 to 10.5 mm.
3 ostia	6.5 × 3.5 to 6 × 9 mm.
2 ostia	8 × 3.5 to 8 × 6 mm.
1 each	9 × 6 and 9 × 8.5 mm.

* One specimen had 2 accessory ostia.

Fig. 6.—Anomalous lateral wall. See text.

ACCESSIBILITY

The larger ostia are especially accessible, while even the majority of the smaller ones are so placed that the antrum can be entered readily through them. The shape may be considered as irregularly round and irregularly ovoid, or a thin slit.

The figures of Schaeffer would seem to be a fair estimate of the probable average incidence of these accessory openings. His figures are based on studies carried out for the special purpose of ascertaining their frequency and are based on the study of 295 maxillary sinuses. He noted 41 per cent of accessory ostia.

UNUSUAL SPECIMEN

In one specimen (no. 17) the infundibulum, the bulla and the uncinate together with the frontal sinus were absent. A fold of mucosa came down from the upper anterior portion of the lateral wall, originating in the region of the posterior border of the lacrimal bone and lay on the lateral wall as a curtain. The anterior part of the curtain merged with the lateral wall, but its posterior portion was a free edge with an opening from behind into the middle meatus. A hooked probe passed from behind, forward and downward fell into a horizontally placed maxillary ostium 2.5 by 3.5 mm. (fig. 6). The middle and posterior ethmoid cells were present. There were no anterior cells.

THE PHYSIOLOGY OF DRAINAGE OF NASAL MUCUS

I. THE FLOW OF THE MUCUS CURRENTS THROUGH THE DRAINAGE SYSTEM OF THE NASAL MUCOSA AND ITS RELATION TO CILIARY ACTIVITY *

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ROCHESTER, MINN.

Biologists and physiologists have been studying ciliary activity for almost a century. Rhinologists seem to have neglected the subject despite the fact that the combined scheme of defense afforded by ciliary activity and the mucinous secretion involves one of the most outstanding physiologic activities of the nose. That the mucinous secretion is an important factor in defense often has been stressed. The part played by ciliary activity has been studied also by a number of writers (Yates,[1] Bloomfield,[2] Bryant,[3] Hartz,[4] Phillips,[5] Schaeffer [6] and Crookshank [7]), but in general it has not been sufficiently emphasized. A comprehensive study of the method and direction of drainage of the nasal surfaces does not seem to have been reported. For studies on the physiology of ciliary movement in general the reader is referred to the works of

* Submitted for publication, June 8, 1931.

* From the Section on Ophthalmology, the Mayo Clinic (work done in the Section on Otolaryngology).

* After a preliminary report of this work had been printed in the Proceedings of the Staff Meeting of the Mayo Clinic (**6**:285, 1931), Dr. A. M. Lucas called my attention to his similar work with Macacus rhesus. An abstract of his report appeared in the Anatomical Record (**48**:27, 1931).

1. Yates, A. L.: Methods of Estimating the Activity of the Ciliary Epithelium Within the Sinuses, J. Laryng. & Otol. **39**:554, 1924.

2. Bloomfield, A. L.: The Fate of Bacteria Introduced into Upper Air Passages, Bull. Johns Hopkins Hosp. **30**:317 (Nov.) 1919; II. B. Coli and Staphylococcus albus, ibid. **31**:14 (Jan.) 1920; The Upper Air Passages as an Environment for Bacterial Growth, Am. Rev. Tuberc. **4**:247 (June) 1920.

3. Bryant, W. S.: An Experiment to Prove That the Cilia of the Human Nose Waft Toward the Anterior Nares, Am. J. Physiol. **33**:430 (March) 1914.

4. Hartz, H. J.: Remarks on the Physiology and Development of the Nose and Accessory Sinuses and Nasal Reflexes, with Special Reference to the Function and Importance of the Turbinated Bodies, Ann. Otol., Rhin. & Laryng. **18**:709 (Dec.) 1909.

5. Phillips, F. L.: The Rôle of Ciliated Epithelium in Sinusitis, Ann. Otol., Rhin. & Laryng. **35**:709 (Sept.) 1926.

6. Schaeffer, J. P.: The Nose, Paranasal Sinuses, Nasolacrimal Passageways, and Olfactory Organ in Man, Philadelphia, P. Blakiston's Son & Co., 1920.

7. Crookshank, F. G.: The Defensive Value of Normal Mucus Formation, and the Theory of Catarrh, Brit. M. J. **2**:627 (Oct. 23) 1920.

Gray,[8] Parker,[9] McDonald,[10] White,[11] Verworn,[12] Grave and Schmitt,[13] Lillie,[14] Maxwell,[15] Carter,[16] Kraft,[17] Pütter,[18] Engelmann[19] and Saguchi.[20]

The present report consists of the results obtained in a study of the flow of nasal secretion of thirty-one persons. The technic consisted simply in dotting small drops of india ink on various portions of the nasal mucosa and observing and recording the direction and rate of flow.

The important part played by the film of secretion on the surface became apparent at once. This secretion extends like a continuous membrane over all the nasal, pharyngeal and esophageal surfaces, from the vestibule of the nose to the stomach. It covers all the surfaces within the sinuses as well as those in the nose proper. This film of mucus is in continuous motion throughout its extent. The direction and rate of motion varies in different regions. The general direction within the

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8. Gray, J.: Ciliary Movements, Cambridge Comparative Physiology, New York, The Macmillan Company, 1928; The Effects of Ions upon Ciliary Movement, Quart. J. Micr. Sc. **64**:345 (March) 1920; The Mechanism of Ciliary Movement, Proc. Roy. Soc., London, s. B. **93**:104, 1922; III. The Effect of Temperature, ibid. **95**:6, 1923-1924; IV. The Relation of Ciliary Activity to Oxygen Consumption, ibid. **96**:95 (May) 1924.

9. Parker, G. H.: The Reversal of Ciliary Movement in Metazoans, Am. J. Physiol. **13**:1 (Feb.) 1905; The Reversal of the Effective Stroke of the Labial Cilia of Sea-Anemones by Organic Substances, ibid. **14**:1 (July) 1905; The Elementary Nervous System, Philadelphia, J. B. Lippincott Company, 1919.

10. McDonald, J. F.; Leisure, C. E., and Lenneman, E. E.: New Principles in the Control of the Activity of Ciliated Epithelium, Tr. Am. Acad. Ophth., 1928, p. 318.

11. White, H. L.: Some Measurements of Ciliary Activity, Am. J. Physiol. **88**:282 (March) 1929.

12. Verworn, Max: Allgemeine Physiologie: Ein Grundriss der Lehre von Leben, ed. 6, Jena, Gustav Fischer, 1915.

13. Grave, Caswell; and Schmitt, F. O.: A Mechanism for the Coordination and Regulation of Ciliary Movement as Revealed by Microdissection and Cytological Studies of Ciliated Cells of Mollusks, J. Morphol. & Physiol. **40**:479 (Sept.) 1925.

14. Lillie, R. S.: The Relation of Ions to Contractile Processes: I. The Action of Salt Solutions on the Ciliated Epithelium of Mytilus Edulis, Am. J. Physiol. **17**:89 (Sept.) 1906.

15. Maxwell, S. S.: The Effect of Salt-Solutions on Ciliary Activity, Am. J. Physiol. **13**:154 (March) 1905.

16. Carter, G. S.: On the Structure and Movements of Latero-Frontal Cilia of the Gills of Mytilus, Proc. Roy. Soc., London, s. B. **96**:115 (March) 1922.

17. Kraft, H.: Zur Physiologie des Flimmerepithels bei Wirbelthieren, Arch. f. d. ges. Physiol. **47**:196, 1890.

18. Pütter, A.: Die Flimmerbewegung, Ergebn. d. Physiol. **2**:1, 1903.

19. Engelmann, T. W.: Zur Anatomie und Physiologie der Flimmerzellen, Arch. f. d. ges. Physiol. **23**:505, 1880.

20. Saguchi, S.: Studies on Ciliated Cells, J. Morphol. & Physiol. **29**:217 (June) 1917.

nose is toward the nasopharynx. The greatest rate is generally found in those areas best protected from the force of inspired air, that is, in the inferior and middle meatuses and in the deeper portions of the common meatus.

The physical characteristics of the secretion suit it admirably to the purposes for which it is used. It is slippery and elastic, and has a certain tenacity. It withstands traction and yet is extremely pliable. It does not readily completely leave a surface under traction, but it can be drawn out in long threads that do not lose tensile strength entirely but are still capable of sustaining traction. It moves with a minimal amount of friction. Its elasticity serves to keep the film spread approximately evenly. The layers of mucin form an effective barrier to the passage of particulate matter and many stains. They entangle and enclose such material, which is then carried away. The ink used in these experiments was carried away without leaving any trace. On the other hand, such solutions as cocaine and ephedrine readily penetrate the film.

The mucinous covering is motivated by several forces: by ciliary activity directly, by gravity, by traction due to cilia and by traction from the pharynx as in swallowing. As the secretion moves away, it is constantly replaced by more secretion produced in the glands contained in the mucous membrane.

In the anterior third of the nose most of the mucous membrane is relatively inactive, as far as ciliary action is concerned. This is the region where the inflowing air first strikes. The extent and distribution of these inactive portions varies greatly, according to individual anatomic characteristics. If deflections and spurs on the septum alter the direction of the currents of air, the inactive areas vary accordingly. They are found where the force of impact of the inflowing air is greatest. Their approximate extent and distribution on the lateral wall and on the septum are indicated by the dotted lines in figure 1 and by the stippled areas in figures 2, 3 and 4. The presence of these inactive areas is stressed for two reasons: First, the drainage from these areas is somewhat different from that of the rest of the nose and sinuses. Second, it is largely on these areas that bacteria, dust, cosmetics and all manner of foreign material contained in inspired air are found to lodge. Drainage of these areas is slow, and is accomplished seemingly by traction on the threads of mucin in the secretion; that is, the secretion lying on the inactive membrane is dragged away by ciliary movement in the adjacent active membrane, as a fish net might be dragged in hand over hand.

DRAINAGE OF ACTIVE AREAS

The regions on the lateral wall, posterior to the stippled areas (fig. 2), drain rapidly by direct ciliary action. They are covered by

ciliated epithelium. The direction of flow of the drops of ink is indicated in figure 2. The general direction is backward, but there is a definite tendency for the direction to incline downward to the margins of the turbinates and thence into the meatuses.

The rate varies, but is usually about 4 to 6 mm. each minute. The time required to traverse the distances indicated is between four and ten minutes. The drops of ink move away en masse. After a few minutes, the entire region is clean. This movement is due to the force of ciliary activity directly on the overlying mucinous secretion. The rôle of gravity is negligible.

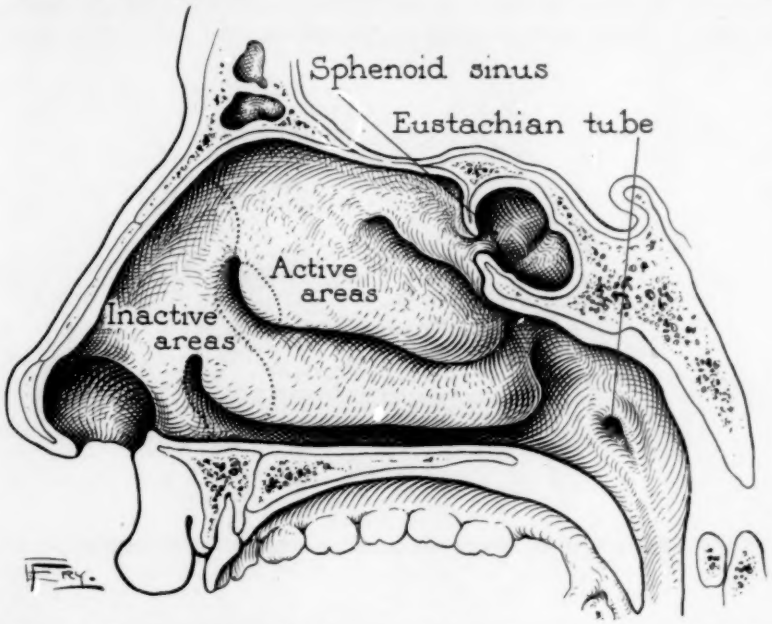

Fig. 1.—The approximate distribution of the inactive areas on the lateral nasal wall.

The two arrows at the vestibule in figure 2 indicate the direction of the flow of moisture condensed from expired air. This usually evaporates in the vestibule.

DRAINAGE OF INACTIVE AREAS

The inactive areas otherwise drain largely through the meatuses as represented in figure 3. The movement is very much slower than that of the active areas. The drops do not move away en masse, except with extreme slowness. Instead, they are drawn away little by little in several fine streaks. Fifteen minutes or a half hour may be required for these streaks to move a few millimeters. When they reach an

active region they speed up and move along to the pharynx in a few minutes. An hour or more may be required for the drop to drain away completely. During this time, there are broken and unbroken black streaks extending from the site where the drop was first placed all the way back to the pharynx. Apparently this drainage from the inactive areas is due almost entirely to traction on the mucus by the cilia farther back. Gravity doubtless exerts some influence on the direction of the streaks, but is not the important factor in the drainage. The greatest ciliary activity seems to be in the meatuses. The secretion travels toward the area of greatest activity. For that reason, it is dragged diagonally into the meatuses, as indicated. A circular area, centering at the anterior attachment of each of the middle and inferior turbinates,

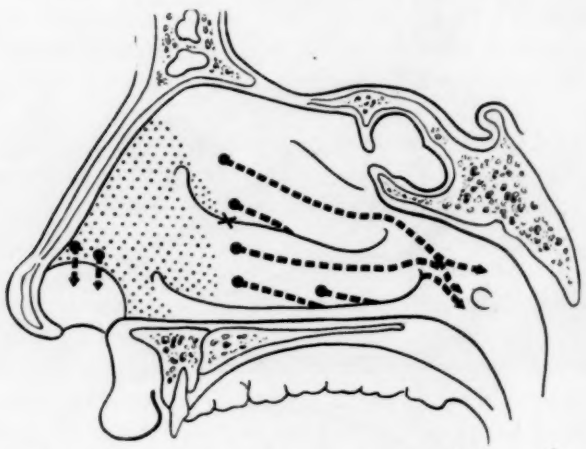

Fig. 2.—The course of flow of mucus on the lateral nasal wall in the active areas.

drains into the meatuses just at the attachment. The small area at the anterior ends of each turbinate, which is included within the circle, drains anteriorly; otherwise the drainage is downward and backward (fig. 3). After passing backward through the meatuses, the ink emerges at the posterior ends and moves on to the lateral pharyngeal wall.

The two types of drainage found and represented in the two sketches (figs. 2 and 3) are very illuminating in the consideration of various bacteriologic studies (Bloomfield and Arnold, Ostrom and Singer [21]). Bacteria sprayed into the nose, as Arnold, Ostrom and Singer sprayed them, would be swept away quickly and completely in a few minutes from the active regions. The inactive areas, although

21. Arnold, L.; Ostrom, M. L., and Singer, C.: Autosterilizing Power of Nasal Mucous Membrane, Proc. Soc. Exper. Biol. & Med. **25**:624 (May) 1928.

lying anteriorly, contaminate the active posterior areas comparatively little since they drain so largely through the meatuses. For that reason, the deeper portions of the nose, between the septum and turbinates, as ordinarily cultured are comparatively sterile. They are continually in the process of becoming sterile. Cultures from the preturbinal region and from the anterior ends of the turbinates might yield, for an hour or two, positive cultures of the organism sprayed into the nose. So might cultures from the middle or inferior meatus and from the lateral pharyngeal wall.[22]

Every rhinologist is familiar with the appearance of the nasal membrane of the patient who powders her nose in the waiting room. If she is admitted immediately after completing her toilet, the powder is

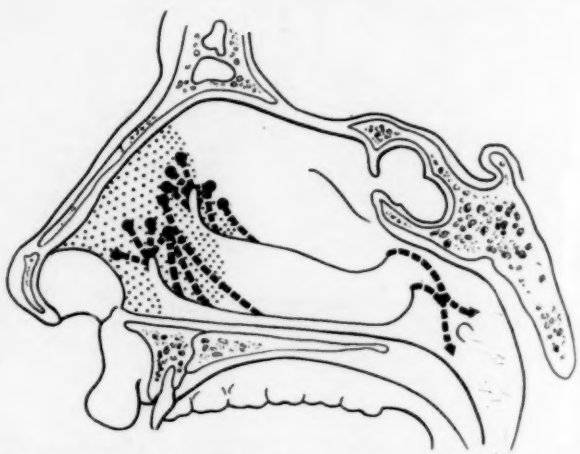

Fig. 3.—The course of flow of mucus on the lateral nasal wall in the inactive areas.

seen to be widely distributed; it is thickest around the anterior end of the middle turbinate and grows progressively thinner as the nasopharynx is approached. If five or ten minutes have elapsed since the last application, there will be no powder visible in the posterior two-thirds, whereas the inactive areas, in the anterior third, will still be thickly covered. The line of demarcation is sharp. If the rhinologist, before applying any medication, takes the time to observe the behavior of the remaining powder he will see it gradually thinned out and drawn into the meatuses, where it disappears.

22. It is understood that this refers to mechanical factors only and omits any consideration of possible destruction by bactericidal substances such as lysozone (Goldsworthy, N. E., and Florey, Howard: Some Properties of Mucus, with Special Reference to Its Antibacterial Functions, Brit. J. Exper. Path. **11**:192 [June] 1930).

The septum, just like the lateral wall, has an inactive anterior zone, where the inspired air first strikes the mucosa (fig. 4). It varies in size and shape according to anatomic variations. On top of a ridge it may extend far back, whereas under an overhanging ridge ciliary activity may be exhibited almost as far anteriorly as the vestibule. The anterior surfaces of spurs are likely to be inactive. Drops of ink dotted on the septum move away to the posterior margin or to the upper surface of the soft palate, as represented in figure 4. The inactive area drains slowly down and back, by traction from the active areas, together with the downward pull of gravity. The direction is the resultant of these two forces. The active areas drain rapidly back toward the posterior margin. There is usually a downward tendency that is slight. Occasionally the ink may be carried somewhat upward, and will emerge

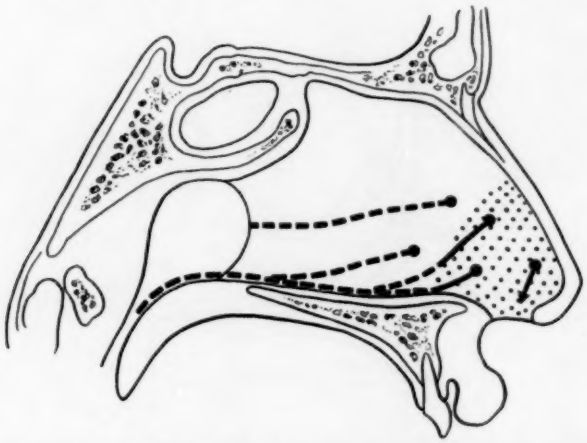

Fig. 4.—The course of flow of mucus on the septum. The rate over the inactive area (stippled) is very slow.

into the pharynx over the rostrum of the sphenoid bone. The fastest rate of travel observed was found in the nose of a patient whose middle turbinate was absent. The posterior margin of the septum could readily be seen. The ink traveled from the anterior limits of the active area to the posterior margin of the septum in three minutes. Ink placed on the floor moves rapidly backward to the upper surface of the soft palate (fig. 5). The inactive zone, anteriorly, is comparatively narrow. The direction is usually almost straight backward. There were some notable exceptions to this. In a few cases, the drainage both from the anterior portion of the septum and from the floor, was directed diagonally across the floor into the inferior meatus, approximately as indicated by the dotted line in figure 5. In at least two of these cases, most of the drainage from the anterior third of the nose, including the septum, floor

and lower half of the lateral wall, was through the inferior meatus. In such cases one should hesitate a great deal before using the mucous membrane of the inferior meatus as a plastic flap to lay in the antrum in operation on this sinus.

When the ink reached the nasopharynx it was soon removed by the act of swallowing. This occurred whether it emerged on the rostrum of the sphenoid bone on the posterior septal margin, on the upper surface of the soft palate or on the lateral wall. The drainage on the lateral wall was backward and downward, dividing about the eustachian tube much as Yates mapped it. Usually it did not proceed very far

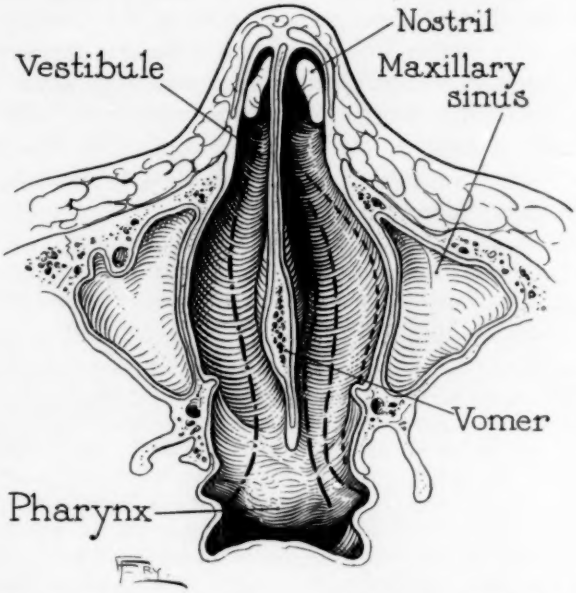

Fig. 5.—The course of flow of mucus on the floor. The line of short dashes represents an unusual direction taken in a few cases. The other lines, although each is drawn in different fashion from the others, are of equal significance and demonstrate the usual course of flow.

before it was entirely disarranged and its progress tremendously hastened by swallowing. It was noted that between swallows the entire lining of secretion within the pharynx slid slowly downward toward the esophagus.

SUMMARY

The nasal mucosa in the anterior portion of the nose, where it meets the inrushing inspired air, does not exhibit ciliary activity, as indicated by the movement of the overlying mucus. This inactive area lies in the anterior third of the nose and drains largely through the inferior and

middle meatuses. It is irregular in size and shape. It ordinarily includes the preturbinal region, the anterior ends of the inferior and middle turbinates, approximately the anterior one fourth to one third of the septum and a narrow strip across the floor. The mucosa posterior to these areas exhibits active ciliary motion, which is greatest in the regions best protected from the flow of inspired air.

The protective layer of mucus that overlies all these nasal surfaces is continuous with that in the pharynx and esophagus. It moves, throughout its extent, very much in the manner of an intact membrane. It is motivated by ciliary action and traction. The nose may be said to have a new lining about once every ten minutes in the posterior two thirds of its extent, and about once an hour in the anterior one third. The drainage of secretion from the anterior inactive areas, where dust and bacteria lodge, is accomplished by traction and gravity. The course from the lateral wall is largely through the meatuses. The motion is slow, requiring an hour or more for drainage. The drainage of the active areas is rapid, requiring only from three to ten minutes. The course in general is toward the nasopharynx.

Clinical Notes

OSSEOUS ATRESIA OF THE POSTERIOR CHOANAE*

W. D. Stinson, M.D., Memphis, Tenn.

A simple and yet comprehensive classification of atresia of the posterior choanae may be made on the basis of the histology and embryology of the atresia, as follows:

1. Membranous:
   (*a*) Lack of rupture of the primitive bucconasal membrane.
   (*b*) Secondary epithelial overgrowths, or epithelial plugs.
2. Osseous:
   (*a*) Bone formation in membranous atresia.
   (*b*) Anomalies of the internal pterygoid plate of the sphenoid bone.

Although it is conceivable that osseous atresia likewise may be due to anomalies of the vomer or posterior septum, I have not encountered or found described such a condition and therefore have not included it in the classification.

Membranous atresia, according to Schaeffer,[1] occurs as follows:

"In the formation of the primitive choanae the dorsal growth or extension of the blind pouch-like primitive nasal fossae meets the ectoderm of the oral fossae. One now finds in these positions the bucconasal membrane composed of two layers of abutting epithelium (nasal and oral) separating the dorsal portion of the primitive nasal fossae from the oral cavity (10 mm. embryos). In 35- to 38-day embryos the membranes are so attenuated that rupture usually ensues, and the primitive choanae are thus established, and with them communication between the nasal fossae and the oral cavity. Lack of rupture of the bucconasal membrane leads to atresia of the choanae, a condition occasionally observed in fetuses and in the newborn. Secondary blocking of previously patent choanal apertures due to epithelial overgrowth has been observed by the writer."

Osseous atresia, as stated before, may occur through the formation of bone in a preexisting membranous atresia, developing either prenatally or postnatally.

Anomalies of the internal pterygoid plate of the sphenoid bone may be explained in two ways. According to Schaeffer, "Coincident with the growth of the facial region and the formation of the palatal processes the primitive or primary choanae become elongated and *form slitlike passageways between the nasal and oral cavities.* The continued growth and ultimate fusion from before backward in the midsagittal plane of the palatal processes in the formation of the secondary palate and the buccal and pharyngeal extension of the primary nasal septum force the primitive choanae to undergo a steady dorsal migration until communication with the cephalic

* Submitted for publication, June 5, 1931.

1. Schaeffer, J. P.: The Nose, Paranasal Sinuses, Naso-Lacrimal Passageways, and Olfactory Organ in Man, Philadelphia, P. Blakiston's Son & Company, 1919, p. 9.

and ventral termination of the pharynx is established. This connection locates the definitive choanae or posterior nares."

The slitlike passageways between the nasal and oral cavities correspond with the bony atresia occasionally found in the adult.

According to Gray,[2] "The internal pterygoid plate has a developmental center separate and distinct from the external plate, those of the external plate appearing about the eighth week, while the internal plate centers do not appear until the fourth month, or the sixteenth week, ossifying in membrane and *becoming joined* to the external pteryoid plate about the sixth month."

Failure of the two plates to join may result in the internal plate being left behind in the lateral development of the pterygoid plate, resulting in the second type of osseous atresia of the choanae.

Membranous atresia is more frequently met with than osseous atresia and is a simple matter to deal with. Osseous occlusion is a more difficult proposition. One of the two cases included in this report was apparently due to ossification of a membranous occlusion, total, bilateral and apparently present at birth, the other to anomalous development of the internal plate of the pterygoid process of the sphenoid bone.

Clinically, the diagnosis is clearcut and obvious, although for some reason it is not generally made. Nasal obstruction with glairy mucus, resulting in dyspnea or cyanosis, in the prone position (if bilateral) and in nursing, should lead one to sound the posterior choanae. Attention should be called to the fact that this syndrome resembles that of enlarged thymus, and is not infrequently diagnosed as such.

REPORT OF CASES

CASE 1.—M. J. M., a girl, was born on March 18, 1927. The delivery was normal. The weight at birth was 7 pounds and 4 ounces (3.3 Kg.). She was a normal child except that from the date of birth she seemed unable to breathe and nurse at the same time. There were no attacks of cyanosis, but she "breathed heavily" during sleep. My first examination was made on October 4, at the age of 7 months. At this time the patient weighed 6 pounds and 1 ounce (2.73 Kg.). The face showed no asymmetry. Closing the nares did not interfere with respiration. The nose was filled with glairy mucus. Sounding the nares showed bony atresia of both posterior choanae. Posterior rhinoscopy was, of course, impossible.

Digital examination (the nasopharynx barely admitting the little finger) revealed a normal contour and size of the choanae, but they were blocked with bone, approximately from 2 to 3 mm. in thickness.

Undoubtedly the method of choice would have been to remove the bony plugs with a small chisel and mallet; however, I was apprehensive of the effect of this procedure on the articulation of the various cephalic bones at this age. Consequently, with the little finger in the nasopharynx, a small sharp-edged Freer submucous elevator was passed through the nose, and the choanae reamed out, much as one would bore a hole through a cigar box with a knife. Hemorrhage was negligible.

Two weeks later, on October 18, the patient had gained 6 pounds (2.7 Kg.), with marked improvement in color and the general condition. The nose was dry, a rubber catheter passing readily through the right naris but with slightly more difficultly through the left.

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2. Gray: Anatomy, New York, Longmans, Green & Company.

On Feb. 14, 1928, there was some scar tissue contraction of the choanae, although they were patent. A strip of gauze was used to "file" away the scar tissue.

After four years, the child is healthy, developing normally, and has few colds. The right choana is of approximately the correct caliber, but the left shows some contraction and should be enlarged. Hearing is normal in both ears.

CASE 2.—A girl, aged 14, was presented for obstruction of the right nostril, which had been present since birth. Examination showed asymmetry of the face. The tonsils were removed. The right naris was filled with glairy mucus; the left was dry and normal. When the left naris was closed, forcible respiration showed only slight patency of the right side. Sounding showed bony occlusion with a small opening at the inferomedian angle of the choana, that is, at the junction of the septum with the palate. Posterior rhinoscopy was unsatisfactory. Digital examination showed the posterior border of the septum to be straight, and the left choana, normal in contour and size. The right nasopharyngeal wall was flat and encroached on the choana. There was a small opening barely admitting a probe at the inferomedian angle. Owing to the asymmetry of the face and the encroaching of the lateral wall of the nasopharynx, the only feasible method was to remove a portion of the vomer, allowing the right naris to communicate with and drain at an angle into the left naris. This was done, with the desired results. The nose became dry and respiration free. Incidentally, hearing was not involved in the right ear.

TRACHEAL ABSCESS

Review of the Literature and Report of a Case *

DAVID H. FAULKNOR, M.D., WATERVLIET, N. Y.

Having found a paucity of references in the literature on tracheal abscess, I wish in this paper to review the literature and to present the report of a case.

In 1913, Richards [1] reported a case of phlegmon which had its origin in a peritonsillar abscess. The patient gave a history of sore throat for several days followed by difficulty in breathing and swelling of the entire left side of the neck. He had paroxysms of dyspnea and coughing. On examination, the tonsillar area was swollen, the patient was cyanotic, and the pulse rate was 124. The skin of the neck was very tense, and there was no fluctuation. The author incised the supratonsillar fossa for abscess. Pus was obtained. At the same time the patient had a paroxysm of coughing. When the opening was enlarged, there was cessation of breathing. An emergency tracheotomy was done, and artificial respiration was produced. During the tracheotomy a phlegmonous area in the neck was entered which had pushed the entire trachea and thyroid area well over to the opposite side, and had itself extended down to a little below the second ring of the trachea. This phlegmon communicated directly with the peritonsillar abscess. There was profuse

* Submitted for publication, July 17, 1931.

1. Richards, George L.: Phlegmon Starting as Peri-Tonsillar Abscess and Extending Downward as Far as the Second Ring of the Trachea, Laryngoscope **23**:835, 1913.

drainage. The temperature dropped to normal. The tracheotomy tube was removed on the eighth day. The patient was discharged as cured on the seventeenth day.

Richard's comment was that the course of the pus probably corresponded to the course of the buccopharyngeal fascia, which invests the constrictor muscles of the pharynx and is given off from the prevertebral fascia, the latter forming its posterior limit. In this case the pus probably forced its way through the fibers of the superior constrictor muscle and thus gained access to the prevertebral fascia.

Another case, that of a man aged 70, was reported by Campbell.[2] This patient had a swelling of the neck for five days. He had a respiratory rate of 32, fetid breath, dyspnea and caries of the teeth. Examination showed marked edema of the aryepiglottic folds, which obstructed the view into the interior of the larynx. Puncture of the edematous fields relieved the edema. A tracheotomy was done, at which time there was an escape of 2 or 3 ounces of brownish watery fluid from the trachea. The patient died thirty-six hours later, apparently of exhaustion.

Campbell's comment on the case was that the patient had an acute laryngeal or tracheal abscess beneath the mucosa, extending from the aryepiglottic folds for an unknown distance down the trachea below the third ring.

Also, in 1922, Theisen,[3] of Albany, N. Y., reported two cases. He made no attempt to cover the literature. He has had the fortune to observe two cases in a period of twenty-five years, which were seen twenty-four years apart.

The first case was that of a man 34 years of age, who had influenza with bronchopneumonia three weeks previous to consulting Dr. Theisen. On examination in the office, there appeared to be a tumor mass in the trachea. His condition was so alarming that he was taken immediately to the hospital, where a tracheotomy was done. The incision through the tracheal wall was followed by a gush of pus and blood. The patient died of bronchopneumonia on the thirteenth day after admission. The other case was that of a man, aged 40, who was also getting over an attack of influenza, but for several days had a cough and difficult breathing. The author was able, under indirect examination, to see a mass which was located at about the third tracheal ring. An emergency tracheotomy was done. The patient did well for four or five days, but died on the eighth day of aspiration pneumonia.

The comment on these cases was that the condition starts as a localized inflammatory process, and then, owing to the patient's run-down condition plus the addition of pyogenic organisms, the abscess gradually develops. The influenza germ was not found on bacterial examination. It is a question whether the lives of the patients with tracheal abscess could have been saved with Jackson tubes and immediate aspiration of pus.

The patient whose case is herewith reported was under my care.

REPORT OF CASE

History.—J. V. A., a white man, an American laborer, complained chiefly of difficulty in breathing and hoarseness.

The father and mother, three brothers and one sister were alive and well. There was no tuberculosis or cancer in the family. The patient, in general, had

2. Campbell, C. A.: Intra-Tracheal Abscess, Laryngoscope **32**:955, 1922.

3. Theisen, C. F.: Abscess of Larynx and Trachea Following Influenza, Albany M. Ann. **43**:505 (Nov.) 1922; Ann. Otol., Rhin. & Laryng. **31**:1118 (Dec.) 1922.

always had good health. He had had tonsillitis every year for the past fifteen years. He had never had rheumatism, chorea or heart trouble. He had typhoid at the age of 11. There was no nasal obstruction or deafness. There was no history of venereal disease. He began to smoke cigarets excessively at the age of 16, continuing until the summer previous to this report, when his family doctor advised against them. There was no history of alcoholism. His average weight was 165 (74.8 Kg.); at the time of his sickness it was 134 pounds (60.8 Kg.).

About Aug. 15, 1929, the patient had acute tonsillitis, which developed into a peritonsillar abscess on the left side. The abscess ruptured spontaneously. He was in bed for about two weeks. Four days after he was up and around the house, he became hoarse. He returned to his family doctor. His subsequent history is that he had remissions and exacerbations to the point of aphonia. He was continuously under his physician's care. He was referred to a throat specialist for treatment. His condition remained unimproved. There were times when he had dysphagia, but there was no embarrassment to respiration. On December 27, I was again called in to see the patient. He had an acute "grip" cold. His temperature was 101 F., the pulse rate 120 and respiration 30. He was dyspneic and cyanotic and coughed spasmodically. He improved slightly until December 31, when he had a sudden closing off of breathing, due to edema of the larynx. His condition became so serious that at 3 a. m. he was removed to the hospital, where an emergency tracheotomy was performed. Oxygen and artificial respiration were required to resuscitate him.

Physical Examination.—Examination showed a tall, emaciated man, who was cyanotic and dyspneic and had an apprehensive expression. The eyes reacted to light and in accommodation; the pupils were widely dilated. The lips and tongue were dry; there was sordes on the teeth and tongue. The tonsils were ragged, cryptic and somewhat red. Laryngoscopic examination revealed a swollen larynx; the left vocal cord was hidden from view by the edema. It was impossible to see into the larynx. There were no diseased teeth. The external surface of the neck presented a phlegmonous area, especially on the left side; no fluctuation could be elicited. There was emphysema all over the neck, extending over the thorax and abdomen. Eleven days after the patient's admission to the hospital, with the aid of a general surgeon, an exploration of the trachea was undertaken. No mass or growth was encountered in the trachea. On examination of the left lateral wall of the trachea, at the level of the cricoid cartilage, a fistulous tract was discovered; probing revealed that this led into a chronic, encapsulated area of inflammatory process extending to the tonsillar region. Pus drained into the trachea after the fistula was enlarged. A counterdrainage was made in the soft parts of the neck, and a light drain was inserted.

There were rough, expiratory breath sounds all over the chest. There were no râles or dulness.

The heart sounds were normal; the rate, 120, regular and rhythmical; the pulmonic second sound was greater than the aortic second sound.

The abdomen was normal, except for emphysematous crackling.

The reflexes were normal.

Laboratory Examination.—The Wassermann reaction was negative on three occasions. The sputum was negative on four repeated examinations. The white blood cell count was 21,000. A smear from the abscess showed Staphylococcus aureus. The roentgenogram of the chest was negative for tuberculosis.

After the abscess cavity was opened and drained, the temperature returned to normal and the pulse and respiration became normal. The patient showed progressive improvement. However, the edema of the larynx subsided gradually. The dysphagia began to clear up, his appetite improved, and he put on weight. He was up and about the hospital on the eighteenth postoperative day; he was permitted to go home on the twenty-eighth day, still wearing the tracheotomy tube. His voice is becoming stronger each day. He is able to breathe fairly well when the tube is corked. A laryngeal stenosis developed, which required that bougies be passed at intervals.

At the time of this report, the patient has gained back his usual weight and strength. The tracheotomy wound has completely healed. His voice is slightly husky, but is improving. Laryngeal examination gives negative results.

CONCLUSIONS

1. In all the cases reported the patients have been men.
2. Staphylococcus is the predominating organism present.
3. Low tracheotomy should be done early when dyspnea develops.
4. The Rose position, or lowering of the head, will facilitate drainage when the trachea is opened.
5. The Jackson tube should be used early in any suspicious case.

825 Nineteenth Street.

Progress in Otolaryngology

A Summary of the Bibliographic Material Available in the Field of Otolaryngology

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FUNCTIONAL EXAMINATION OF HEARING; DEAF-MUTISM AND EDUCATION OF THE DEAF *

ROBERT SONNENSCHEIN, M.D.
CHICAGO

In the review of the literature on the functional testing of hearing published in the January, 1930, number of the ARCHIVES,[1] it was stated that one of the most striking facts in the period from Oct. 1, 1928, to the end of September, 1929, was that so few articles had appeared in the foreign literature, especially the Continental European literature. "This is a decided reversal of form from that which was observed in previous years when American or English writers seemed to deal with this subject only on rare occasions." The same thing may be said this year, that not only are there fewer articles altogether, but they are practically all by English and American writers, particularly the latter. It is difficult indeed in so limited a sphere and one that has been studied so intensively to bring forth new material, and I believe, therefore, that great credit is due our American colleagues for their contributions to the literature of the subject the past year.

Froeschels [2] stated that he and Tullio independently of one another demonstrated an eye muscle reflex produced by noises. Tullio used animals and in some cases human beings, testing them in the dark, while Froeschels tried it on so-called deaf-mutes. Tullio described his method as that of the conduction of a very intense sound to the ear of pigeons by means of tubing, and noting the turning of the head about the occipitofrontal axis. In these cases, as a rule, the head was turned to the side opposite the one to which the sound was conducted. In mammals, not only were there movements of the head but analogous movements of the eyes. When a single semicircular canal of the animal was injured, the reflex movements were in the plane of that canal, and when several were injured, the movements were in the resultant plane.

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1. Sonnenschein, Robert: Functional Examination of Hearing; Deaf-Mutism and Education of the Deaf, Arch. Otolaryng. **11**:97 (Jan.) 1930.

2. Froeschels, Emil: Eye Muscle Reflex Produced by Noises in Cases of Advanced Hearing Impairment, Ztschr. f. Hals-, Nasen- u. Ohrenh. **26**:511, 1930.

Tullio also showed, in his demonstrations on man, with fixation on a point of light in an otherwise absolutely dark place, movements of the eyes occur when a vibrating tuning fork is either placed on the skull or held in close proximity to the ear.

Froeschels,[3] in 1923, devised a new method for the testing of hearing of the "so-called deaf-mutes" who very often have merely greatly impaired hearing. This test consisted of placing an otoscope tube in each ear and conducting through it the tones of an Urbantschitsch harmonica for the purpose of determining whether any remnants of hearing remained. During the examination he noted movements of the body and jerky movements of the eyes (both as a rule but in some only one). This was not due to the presence of a fistula symptom, because blowing in of air without sounds does produce movement of the eyeballs. The direction of the eye movements was usually away from the ear that was being stimulated. In many cases the plane of the movement was horizontal, in others rotatory and upward, in some obliquely upward and occasionally vertical. In some instances one tone would cause a movement in a different direction from that produced by another tone. It was often noted that the movement produced by sounds in the better ear was more definite than that from the less hearing ear. Froeschels therefore came to the conclusion that the better ear was more easily stimulated than the worse one. He expressed the belief that the reflex movements of the eyes, head and body depending on auditory stimuli were probably characteristic of man in previous ages, so that their recurrence in cases of markedly impaired hearing points to the rarity with which movements of acoustic stimulation are seen and is in these cases evidence of an atavistic tendency.

Macfarlan [4] discussed the speech test, its shortcomings and the steps taken toward remedying them. The phonograph speech test with mechanical control of the intensity has been a great improvement. Attention, alertness, eagerness, memory, general intelligence, etc., play an important part in all tests of hearing, especially in the speech tests. However, little has been developed concerning these psychologic aspects of the tests of hearing which become evident when the deaf child is brought to the otologist for examination and diagnosis. These children usually have little or no speech. The questions that arise in these cases are: Were they born deaf; is the hearing impairment partial or complete; is the altered psychology due to the deafness or is it evidence of mental stigmas? The question of the hearing may require several visits before the apprehension of the patient is overcome and his auditory

3. Froeschels, Emil: An Eye Muscle Reflex in Deaf-Mutes Elicited by Noises, Monatschr. f. Ohrenh. **61**:776 (July) 1927.

4. Macfarlan, Douglas: The Status of Tests of Hearing, Arch. Otolaryng. **13**:47 (Jan.) 1931.

attention and responses have been trained. At times it is very difficult to differentiate between tactile and auditory response. This can be determined by placing the handle of a sounding fork on the mastoid, the patient moving his hand as long as he hears it. When the hand is stopped, the fork handle is inserted into the external auditory meatus, and if hearing is present, the patient will again raise his hand. It may be impossible at times to elicit response to tests for single tones until the familiarity and interest are developed in the patient, after which these tests may be used with favorable results. The use of the 4-*A* audiometer record of speech numbers in these cases is absurd as these children have never used language. Their degree of loss of hearing can be roughly estimated if a record of simple rhythm is used, amplified through two tubes and controlled by an audibility meter which reads the loss of sensation units.

In children and adults who have had hearing and who know language, speech may be used as a testing stimulus. It is important to use words of common knowledge, familiarity and experience. Macfarlan has prepared a list of such words which is printed. Selected words from this list have been taken and made into a record. The 4-*A* audiometer is used, and the child recites the words heard instead of writing them. Recitation permits more attention to hearing. The examiner writes down the responses and compares them with the key. This method has been found useful in testing the preschool child. The intensity of the record may be gradually reduced to the point where the child begins to miss, and this is considered his hearing threshold. The importance of small loss of hearing is being appreciated more as time goes on. A loss of hearing for speech of from 45 to 50 per cent means that a person cannot hear or understand speech and cannot be helped by aids of hearing. A loss of from 18 to 20 per cent signifies a severe handicap in understanding voices at conversational distance, about from 6 to 9 feet. The patient with a loss of 18 per cent will invariably show the developing psychosis of deafness. The smallest degree of loss, from 6 to 9 per cent, causes a child to have difficulty with his school work.

Becker [5] presented a simple device which he believes serves the purpose of testing the auditory function of each ear separately by bone conduction. This can be accomplished by ruling out one labyrinth while testing the other as in the air conduction test, the Bárány instrument being used. He found that when a stream of air is directed steadily and forcefully against the tympanic membrane all sounds by bone conduction are eliminated as well as those sounds used in air con-

5. Becker, B. M.: Testing for Perceptive Deafness, Laryngoscope **41**:246 (April) 1931.

duction tests, except for the screaming voice at the ear and the upper limits of the Galton whistle. This is accomplished by placing one end of a rubber tube with an ear tip loosely in the ear to be eliminated, and blowing a forceful current of air directed against the drum while a strongly vibrating fork is placed over the mastoid antrum of the ear under examination. Care should be taken that a free return of air from the canal is obtained. "If the patient hears the sound of the fork there is no doubt that he hears it on the side under test. If he does not hear it, we are reasonably certain that there is a complete loss of function or a grave functional condition of the cochlear portion of the labyrinth tested."

I do not see how this method of using a noise apparatus actually tells whether the patient hears via mastoid of the tested ear or via the opposite ear by diagonal resonance or crossed perception. The latter subject has been discussed by me in reviewing a number of articles in previous January issues of the ARCHIVES.

Shambaugh [6] presented interesting statistics on deaf-mutism and hearing impairment in children. There are approximately 45,000 mutes in the United States. This includes only those who acquired deafness before the age of 8. Approximately 10 per cent of the school children suffer from some defect in hearing. There are about 3,000,000 children in the United States with subnormal hearing and over 10,000,000 with some defect in hearing. The important step in the prevention of congenital deafness is to discourage consanguinity, as well as the bearing of children, by those with a family history of congenital deafness. The etiology of acquired deafness is mainly meningitis, first, measles second, scarlet fever and influenza third. Hereditary syphilis does not appear to be an important factor in the etiology of severe deafness in children. From the study of acquired deafness, it was shown that little can be accomplished in the prevention of profound deafness from infectious diseases by more prompt or better care of the otitis media complicating these diseases. The only hope lies in the prevention and the prompt cure of the disease itself. Periodic examination of the ears of school children is very important. This may aid in preventing progressive deafness in adults by relieving and correcting tubal trouble in children. The audiometer from the practical standpoint is not necessary. It serves to divert the attention from the practical objectives in addition to being more or less troublesome. The tangible objectives are most easily obtained by employing the simple voice tests.

The practical proposal for the prevention of otosclerosis is to discourage propagation by those with a family history of that disease.

6. Shambaugh, George E.: The Problems of Deafness, Arch. Otolaryng. **14**:36 (July) 1931.

The problem of readjustment is great. Lip reading seems to be the most important aid to persons with progressive deafness. Mechanical devices such as aids to hearing take a second place. The problem of rehabilitation for those who have lost their hearing in adult life has been aided considerably through the efforts of Dr. Wendell Phillips and the League for the Hard of Hearing. A plea is made for all to aid in the problems of rehabilitation and human salvage of the deafened which have been greatly neglected.

This timely paper emphasizes many important points in the distressing problem of impaired hearing.

Bunch and Raiford [7] made a study of the variations in auditory acuity in males and females, and in white and colored patients. These tests were made on patients from the medical and surgical departments of the Johns Hopkins Hospital, and the averages of the thresholds of auditory acuity secured by audiometric measurements were computed according to age. They found the average acuity for tones under 2,048 double vibrations slightly higher in white males than in white females. Near 2,048 double vibrations the averages for the two sexes cross, and the average for females is higher than for males. This is true for all decades. There is no definite difference between colored males and females. The average for colored males is higher than that for white males for tones of high pitch. "In each successive decade the average of the threshold is slightly lower for tones of low pitch. For tones of high pitch the difference is more marked than for low pitched tones, and the difference between the average for each age group is greater as the frequency of the tones used as stimuli increases."

Bunch [8] continued his work on auditory acuity in the group of 468 patients who have arteriosclerosis, hypertension, chronic cardiac conditions, syphilitic and various malignant conditions to determine their effect on auditory acuity. In an earlier study, he found that a loss in acuity for tones of high pitch with advancing age is a constant factor in patients having the aforementioned diseases. However, no one of these systemic conditions can be considered responsible for this loss.

Arteriosclerosis is said to be a constant factor which increases with advancing age. If this is true, then it parallels the loss shown in this study and in the one previously presented. The observations in the present study seem opposed to this contention, as patients in all decades for whom a definite clinical diagnosis of arteriosclerosis has been made do not show a greater loss than those for whom such a diagnosis was not made.

7. Bunch, C. C., and Raiford, T. S.: Race and Sex Variations in Auditory Acuity, Arch. Otolaryng. **13**:423 (March) 1931.

8. Bunch, C. C.: Further Observations on Age Variations in Auditory Acuity, Arch. Otolaryng. **13**:170 (Feb.) 1931.

Josephson,[9] as a result of careful observation, stated:

> There exists a definite type of deafness for low tones associated with retraction of the tympanic membrane which will not respond to inflation and which is not associated with any gross pathologic process of the mechanism of the middle ear. Both the deafness for low tones and the retraction of the tympanic membrane are relieved by galvanization of the ear. The improvement is due to relief of the spasm of the muscles of the accommodative mechanism of the middle ear.

I have had no experience with this subject, but hope others will be able to publish work in reference to confirmation of Josephson's rather striking communication.

Wilder[10] noticed in the study of labyrinthine deafness with the tuning forks that with a total loss of air conduction in one ear, and the Weber test lateralized to the well ear, when the tuning fork is placed over the affected mastoid the patient frequently claims he perceives the sound with the diseased ear. In order to test whether or not this bone conduction is "borrowed," an artificial total obstruction is created on the affected side by closing the canal with the finger, while the tuning fork (256 double vibrations) is placed on the mastoid of the affected ear. If one is testing a labyrinthine deafness and a dead ear, the patient does not notice any difference in the intensity of the sound, but if some hearing remains on that side the sound is greatly increased.

As a check on this observation, the finger is removed from the affected ear and the canal of the well ear is closed. The patient immediately will state that he notices a difference in the intensity of the sound in the unaffected ear. The fork is then placed over the mastoid of the unaffected ear. When the finger is placed in the canal of the diseased ear, the patient should notice no difference in the sound, whereas closure of the canal of the unaffected ear will greatly magnify it.

By means of the test one can be practically certain that all the bone conduction is being heard by the unaffected ear. Bárány tests would also help to establish the diagnosis.

The principle of one of the types of the Bing test is that employed in Wilder's work and serves as a substantial aid.

Pohlman's[11] observations are opposed to the accepted view that the apparatus for the transmission of sound is functionally more efficient in the lower frequency range. He found the drum membrane and the ossicular chain of nearly equal functional efficiency throughout the pitch range by comparing average acuity curves for air and bone conduction

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9. Josephson, E. M.: Spasm of the Auditory Accommodative Mechanism, Arch. Otolaryng. **13**:166 (Feb.) 1931.

10. Wilder, Henry S.: A New Test for Borrowed Bone Conduction, Arch. Otolaryng. **13**:822 (June) 1931.

11. Pohlman, A. G.: Interpretation of Conduction Deafness, Arch. Otolaryng. **14**:48 (July) 1931.

of normal persons with those persons giving a negative reaction to the Rinne test with obvious fixation of the stapes but normal acuity of bone conduction. This information was directly correlated with the acoustic insulation of the end-organ, and with the efficiency of the apparatus for the transmission of sound reported in a previous article. If this is true, three possibilities present themselves: first, that the accepted observations in these cases are not real phenomena; second, that the accepted results of the functional tests in conduction deafness must be interpreted on some other basis than variable efficiency in the apparatus for the transmission of sound, and finally, the possibility that the evidence in favor of equal functional efficiency of the apparatus for the transmission of sound throughout the usual pitch range is at fault.

As to the realness or the unrealness of this phenomenon, the same explanation may apply here as in the cases of paracusis willisii, which has been shown not to be a real phenomenon. In comparing his hearing with a group of patients with negative reactions to the Rinne test, Pohlman's hearing was less acute above 4,000 double vibrations than the patients'; but reversed below 2,048 double vibrations. This does not mean the patients' transmission apparatus showed a greater loss at the low frequency end of the audible range, but demonstrates that the writer was distinctly presbycusic. As long as one hears the lower frequency range at normal intensity, then one's sound and perceptive mechanism may be considered as relatively normal. If the apparatus for the transmission of sound is equally efficient at all frequencies, then the person should hear equally well at all frequencies, and if he does not, one assumes a perception deafness which should register both by air and by bone conduction. This was the case with Pohlman's hearing. A person with normal hearing will hear all frequencies better with normal perception apparatus than one with conduction deafness. The more pronounced the presbycusis in the examiner, the more pronounced the low pitch disability in patients with conduction deafness, and the phenomenon is not real.

Regarding the second possibility as to whether this is a real phenomenon, it is true that persons with conduction deafness with an equal depression in the acuity of hearing for the entire pitch range show greater disability at the low frequency end of the audible range. These persons also show a greater loss at the upper end of the audible scale than in the region of the intermediate frequencies. This was first established by Wegel in 1922.[12]

This result is dependent on the fact that the region of greatest sensitivity in the internal ear is at the same time that with the greatest range in intensity. The

12. Wegel, R. L.: The Physical Examination of Hearing and Binaural Aids for the Deaf, Proc. Nat. Acad. Sc. **8**:155, 1922.

result is therefore a real phenomenon that has nothing to do with the efficiency of the apparatus for the transmission of sound.

However, the interpretation in terms of function of the middle ear has been erroneous.

Pohlman presented two unusual cases with a complete discussion to substantiate his conclusions which are as follows:

1. The apparatus for the transmission of sound is about equally efficient in the transmission of all audio-frequencies to the cochlea.

2. The usually accepted interpretation that conduction deafness results in greater losses in acuity for hearing low pitch tones because the apparatus for the transmission of sound is more efficient at the lower end of the pitch range is incorrect.

3. The fact that relatively greater losses in acuity occur at the lower end of the frequency range is dependent on an equal efficiency in the apparatus for the transmission of sound and on the correlation between the range of sensitivity and intensity at the various levels of the perceptive apparatus. The conduction deafness, therefore, merely brings to consciousness the normal reaction of the internal ear itself.

While Pohlman is probably perfectly correct in his assertion regarding the fact that the apparatus of transmission of sound is about equally efficient in the transmission of all frequencies when this work is done with the audiometer, one must remember that in practical tests such as the voice, etc., this apparently is not true. It is quite true as I have pointed out a number of times, that voice tests cannot be actually standardized, but when certain precautions are taken, which I will not again repeat here, it is possible to get a very good idea of the actual state of a person's hearing. When unaccentuated whisper is used, one often finds that the patient hears the low pitched tones at a great distance, but the high ones greatly reduced. In these cases one knows that there is a beginning change in the perception apparatus. On the other hand, when the high pitched sounds are heard at normal distance, but the low ones are heard less far, it usually means a beginning conduction impairment. The very high tones are not heard by many people, and since they are above the range used in conversation, one is not always aware of the fact as to whether one does or does not hear them. Therefore, even though Dr. Pohlman's conclusion is entirely correct from a scientific standpoint, one should remember that clinical findings are the criterion in making a diagnosis and determining a prognosis. The classic work of Bezold should be emphasized. Many autopsies verified the pathologic changes he diagnosed with the standard voice and tuning fork tests.

Knudsen and Jones [13] bring to mind the present concept of paracusis, which is that no one hears better in the presence of a noise. A para-

13. Knudsen, Vern O., and Jones, Isaac H.: Bone Conduction, Arch. Otolaryng. **13**:489 (April) 1931.

cusic person has relatively better hearing than a normal person in the presence of noise because he is not bothered by the noise, and does not elevate his voice as a person with normal hearing does under similar circumstances. The apparent increased bone conduction found in patients with fixation of the stapes and chronic catarrhal otitis media when tested in one's office is found to be lacking when the patient is tested in a quiet place. This is due to the masking effect of the noise in the office. However, an actual increased bone conduction is found in an ear having the external canal occluded with a plug of wax or water. No theory has as yet been evolved to explain this. Knudsen and Jones attempted to establish a normal standard for bone conduction for tones of different pitch and to compare this with that found in pathologic cases, testing both in the office and in a sound-proof room. Bone conduction measurements with tuning forks are limited to rather low frequencies since the damping of high frequency forks is so great as to render their tones inaudible before or shortly after the forks are placed on the skull. A bone conduction receiver such as is used in audiometric measurements is satisfactory because the vibrations can be maintained at a definite amplitude and varied by an ordinary volume control. In addition, a tone of any desired pitch and loudness can be maintained. The special bone conduction receiver used for these tests had an aluminum rod 6 inches long attached to a narrow strip of magnetic steel placed directly across the poles of the receiver. The free vibration of the steel strip is communicated to the rod, the distal end of which is pressed firmly against the head. The sensitivity of hearing by bone conduction was measured by determining the amount of vibration of the rod when the bone transmitted sound was reduced to the threshold of audibility. Bone conduction can readily be measured in a sound-proof room for frequencies between 64 and 4,096 double vibrations by adjusting the volume control of the bone conduction receiver until the tone becomes barely audible. The readings on the dial can be regarded as normal. These can then be compared with readings obtained in cases with impaired hearing acuity. The minimal threshold for bone conduction can be tested as high as 4,096 double vibrations, but the amplitude of the rod was not obtained above 1,024 double vibrations.

The authors found that the amplitude of vibration was very small, decreasing as the frequency increases. The corresponding amplitudes of vibration of the drum membrane at the minimal threshold for air conduction are of the order of one one-hundredth of the observed amplitude of vibration of the aluminum rod in the measurements of bone conduction. So in order to communicate sound to the cochlea by means of a bone conduction receiver making contact with the mastoid process, the amplitude of vibration communicated to the skull must be about one hundred times greater than that communicated to the drum

membrane of a normal person by air conduction. When curves of both air and bone conduction are plotted and compared, the sensitivity of the ear seems to vary in about the same manner for both bone and air conducted sounds. The sensitivity increases for both in about the same proportion from 128 to 1,024 double vibrations. The amplitude of vibration at 128 double vibrations for both is fifty times larger than the corresponding amplitude at 1,024 double vibrations. This suggests that the variation in the sensitivity of hearing is primarily a function of the properties of the cochlea rather than the mechanical properties of the conduction mechanism.

The acuity by bone conduction for normal persons was found to be higher in the sound-proof room than in the office. A group of persons with pathologic conditions was tested, and it was found that they showed no actual increase in bone conduction. These conditions included otosclerosis, chronic catarrhal otitis media, trauma of the middle ear, etc. This simply indicates that types of hearing impairment with which one associates increased bone conduction do not show it when tested in a sound-proof room. Charting both the percentage of hearing by bone conduction and air conduction on the same graph aids in making a diagnosis. Quantitative measurement of bone conduction over a wide range of frequencies facilitates differential diagnosis, enabling one to determine the degree to which either the conductive or the perceptive lesion has contributed to the lesion. Knudsen and Jones believe testing of the hearing in a sound-proof room is advantageous, and present a simple plan of construction of such a room.

Work in the silent chamber is certainly of very great value, for with the noises usually present in the office and elsewhere eliminated, the true picture of the actual bone conduction is obtained. From a practical standpoint, however, one must say that it is not possible for every otologist to have a sound-proof room, and therefore, finding apparently diminished prolonged bone conduction will still serve as a criterion in making diagnosis of a functional test. If the tests are always made in the same room, with the same furniture, so that the sound pattern, or rather the wave pattern in the room is always the same, one gets results that one can compare with other findings in order to see if the particular patient's bone conduction is apparently normal, diminished or prolonged. However, rather than have the examiner use his own bone conduction to compare with that of the patient, it is much better to have an objective standard. As I have so often stated, this can be obtained by using one fork in the same manner on a large number of apparently normal persons, and then comparing the average figure thus obtained with the findings in the patient. The results are comparable, even though from a strictly scientific standpoint the tests would be

more accurate if they could all be done in a properly constructed soundproof room. I am a firm believer in accuracy and in scientific methods, but some of them do not always lend themselves to conditions under which the overwhelming majority of otologists must do their work.

Pohlman [14] stated that patients with conduction deafness hear as normal persons do, provided sufficient intensity is developed to compensate for the lowered acuity. As a result, these patients hear telephone conversation well because the telephone output lies beyond the maximum possible disability due to the functional loss of the sound transmission apparatus, which is about 40 double vibrations. The auricle acts as a reenforcing agent for sounds of the high frequency end of the audible range. This explains the increased efficiency shown in perception deafened persons when the hand is cupped back of the ear. The acoustic fan in cases with stapes fixation in which the Rinne test is negative may be of great benefit because the air sound transmission apparatus in these cases is not functioning, enabling the person to appreciate the contribution to the intensity developed in the internal ear. However, the fan advertises the disability and also prevents speech while the patient is listening at the same time, which are two objections to this apparatus.

Speech may be transmitted to the cochlea either by way of the air sounds or by that of bone sounds, but not by both. The spoken whispered test has a number of serious disadvantages which apparently are not fully appreciated. There is the variability of voice intensity and production, intelligibility of the various speakers and character of the room in which the test is made. Subjectively, the intelligence of the patient, attention and familiarity with the material spoken in testing are factors. Also, the inverse square law governing the intensity does not hold good and is directly affected by a number of factors.

The spoken voice, on the other hand, can be used in such a manner that the quality is standard and the intensity held under a quantitative control. Pohlman showed that the air sound transmission apparatus may be regarded as equally efficient throughout the audible range. This applies to bone transmission also. In view of this it was thought possible to correlate the acuity for hearing both air and bone transmission quantitatively in spite of the different routes taken by the two types of vibrations. This was accomplished by a method devised at the audiometric laboratory of Dr. J. F. Fairbairn of Buffalo. An air telephone was damped and desensitized so that the peak response coincides with that of the bone telephone when placed under the same conditions of amplification and attenuation. If the two transmission routes are about equally efficient and the two telephones are also of

14. Pohlman, A. G.: The Possibilities in the Quantitative Correlation Between Air and Bone-Transmitted Speech, Laryngoscope **41**:157 (March) 1931.

equal efficiency throughout the range, then the acuity curves should coincide in the same persons within a reasonable error. Several graphs are shown for various defects. The results of these correlations indicate there is no evidence of a mechanical selectivity in reference to the frequencies transmitted for either air or bone sounds. This method may be applied to a direct quantitative correlation between the acuity for hearing air and bone transmitted speech provided the two types of receivers are definitely evaluated on the basis of quantitative responses. These possibilities are now being considered, a Western Electric 4-*A* audiometer with the electromagnetic pickup being used as the source, modified so that the maximum output available from the spoken record (75 double vibrations) is available for bone conduction.

I believe it is a long accepted fact that air-borne sounds such as the voice, etc., are heard by both air and bone conduction. When the sounds are generated in the air one hears mostly by air conduction via the external auditory canal, and little via the eustachian tube, but only to a moderate degree by bone conduction, that is, by way of the sound waves that strike the skull.

Pohlman [15] presented a comprehensive discussion on transmission of sound by air and bone conduction. So many essential points are mentioned in this excellent study that the paper does not well lend itself to abstracting. Therefore, only a portion of the many conclusions are here cited. The reader should peruse the whole article for a better appreciation of the problem presented. Portions of the author's summary are given here:

1. The auditory end organ may be regarded as acoustically insulated against air vibrations because it is immersed in the liquid of the internal ear.

2. The middle ear apparatus may be considered to have the function of a mechanical transformer which matches the impedance differences between air and water in the transmission of vibrations.

4. The efficiency of the air sound transmission system is so great, when compared with that of the direct transmission of air sounds through the medial tympanic wall (round window), that the contributions through the latter to the intensity of the signal developed in the internal ear through the former are too small to come to consciousness. The two routes are, therefore, not simultaneously operative in a physiologic sense.

5. Where the sound transmission apparatus is at all functional, there the case tends to report Rinne positive, and not until the drum membrane and ossicular chain route is functionally out of commission does a definite Rinne negative condition obtain (Riverbank magnesium alloy fork 256 Hertz). This is true except in cases where the drum membrane is loaded or where the external canal is occluded.

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15. Pohlman, A. G.: Correlations Between the Acuity for Hearing Air and Bone Transmitted Sounds in Rinne Negative and Rinne Positive Cases, Ann. Otol. Rhin. & Laryng. **39**:927, 1930.

7. Bone sounds are transmitted to the cochlea either through the wall of the otic capsule (cranial) or by way of the drum membrane and ossicular chain (craniotympanic). Under conditions where direct bone transmission is effective, the contributions to the intensity developed in the cochlea through additions by way of the drum membrane and ossicular chain do not appear to be sufficient to influence the threshold stimulus. Accordingly, the bone acuity curve is not influenced by the functional condition of the air sound transmission system, except as noted above, and is independent of the Rinne positive and Rinne negative report.

13. The acuity for hearing bone sounds is far more variable than that for hearing air sounds, even under conditions of a test chamber. This must always be borne in mind in the differential diagnosis. The masking effect on bone sounds, particularly those of low frequencies, of even slight adventitious noises, cannot be overemphasized and for accurate tests a silence room is quite essential for all Rinne positive cases.

14. Lateralization of bone sounds occur:

(a) To the side of greater sensitivity in the perceptive apparatus (direct cranial transmission).

(b) To the side where the drum membrane is loaded or the external auditory canal is occluded (craniotympanic or prolonged bone transmission).

(c) To the side of conduction deafness, where the effect is due to masking of the better ear (false lateralization).

15. The correlations between the air and bone acuity curves obtained by audiometric methods are quite in agreement with those obtained through fork tests except in conduction deafness. Here the differences are explained by variations in the intensity of air and bone sound delivered by various forks; by the greater minimum-maximum sensitivity of the internal ear in the upper fork range; by the masking effect of office noises; and by the degree of presbyacusia in the examiner.

Mundt [16] stated that the audiometer is the only mechanism by which a reasonably accurate percentage hearing loss may be determined, and that it is a practical instrument for making graphic records and keeping an accurate account of the progress of the patient. This progress can also be watched by different otologists. It is more satisfactory in patients who will not cooperate sufficiently for good fork tests, especially in children. It is the best means for testing the upper portion of the scale, but not as efficient as other methods in testing the lower tones. Mundt expressed the belief that only a very small proportion of otologists are equipped mentally and physically to make satisfactory fork tests, and that many competent men are not willing to procure or use forks or whistles acceptable for good work.

I believe it is easy for otologists to learn to use a few good forks properly, and thus to make the fundamental tests satisfactorily. These with examination of the ear, nose and throat, testing with the voice and the evaluation of the history of the case enable one to make the diagnosis.

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16. Mundt, G. Henry: The Practical Value of the Audiometer, Ann. Otol., Rhin. & Laryng. **40**:215 (March) 1931.

Drury [17] examined 126 cases of reported ear involvement associated with hepatic dysfunction. The clinical syndrome included a series of symptoms, the most common of which were deafness, tinnitus, vertigo, headaches, fatigue and nausea. The characteristic audiometer findings seemed to be a drop at 4,096 double vibrations. This syndrome is supposed to be due to a long-standing toxemia arising from a disarrangement of the liver chemistry and is said to be greatly relieved by proper care and treatment.

This subject is most interesting and warrants much further study, before definite conclusions can be reached.

Fowler,[18] in response to many requests, gives a thorough, concise explanation of the audiogram, what it is and what it means. The audiogram is a graphic representation of hearing capacity plotted in sensation units, each unit being approximately the amount of increase or decrease in the power or energy producing a sound just sufficient to be appreciated as a change in loudness. This unit is not only a logarithmic measure of the loudness of sound, but also a measure of change in sound intensity. For each frequency of the tonal range a given amount of sound pressure is necessary so that the normal ear may detect its presence, the minimum amount necessary being at about the middle of the tonal scale and maximum amounts at both ends. Therefore, in approaching the higher and lower tones, a point is reached where the ear cannot differentiate between hearing and feeling. These points are known as the upper and lower limits of hearing, respectively. The audiogram can be made by any instrument, such as a tuning fork, whistle, audiometer, etc., if properly calibrated for time or distance. The reason the audiometer cannot be heard in the higher tones as from 11,585 to 16,384 whereas the Galton whistle is heard at these frequencies, is that the Galton whistle is many times louder and has at least four different sounds some of which are quite low. If the Galton whistle could be calibrated for intensity with the elimination of its adventitious sounds, one would find that both the audiometer and the whistle would give similar readings in similar cases.

> Unless tuning forks are carefully calibrated, no two users can quantitatively compare their results. . . . The greatest defect in the tuning fork is its inability to maintain a fixed intensity so that it is difficult to obtain as accurate a threshold of hearing as with the audiometer with which any intensity may be repeatedly applied to the ear.

Fowler impresses one with the difficulty of interpretation of the audiogram in some cases from a diagnostic standpoint and illustrates

17. Drury, Dana W.: An Aural Syndrome of Hepatic Origin, Ann. Otol., Rhin. & Laryng. **40**:415 (June) 1931.

18. Fowler, Edmund Prince: Interpretation of Audiograms, Arch. Otolaryng. **12**:760 (Dec.) 1930.

this with four cases in which both the audiometer and tuning forks were used. He shows the necessity of testing the hearing by air conduction at all frequencies, using the tuning forks, Galton whistle or the audiometer, and also testing bone conduction at several frequencies up to 4,096 double vibrations in order to assure an accurate diagnosis.

Ersner and Pressman [19] presented an interesting study in attempting to prove the assumption that under certain conditions the audiometric tracing gives a false picture of air conduction in that it is also recording sound transmitted through bone. This phenomenon can be recognized only in cases in which the reaction to the Rinne test is negative, for otherwise the higher level of the air curve obscures any element of bone conduction that might be taking place. They believe that when testing with the audiometer ear phones in cases in which the Rinne test is negative, the true proportion that bone conduction plays in audition is greatly exaggerated. In view of the contact that exists between the ear phone and auricle, it is not unusual to expect the occurrence of an undue amount of bone conduction. They cite Sonnenschein, who stated that a fork placed over the tragus conducts not only by air but by bone and cartilage. Such a contact occurs in an exaggerated form when the telephone receiver is used. However, they disagree with Macfarlan and Fletcher, who deny an undue element of bone conduction in audiometry on the basis that the sounds produced are transverse and not longitudinal vibrations, as they feel the weight of experimental evidence tends to disprove them.

Ersner and Pressman found that in persons with a negative reaction to the Rinne test with marked differences in ability to hear conversational voice and tuning forks, the audiometer curves were very similar. Because of the great similarity of the air and bone conduction curves obtained in these cases, they believed the discrepancy might be due to bone conduction taking place through the ear phone. They conducted several experiments to determine this further. In one series of cases in which bone conduction tested by the fork was normal and considerably better than air conduction, the air and bone conduction curves were nearly similar. This was interpreted as being due to the ear phone giving rise to bone conduction and recording it rather than the air conduction. They also found that by increasing bone conduction in the opposite or normal ear by packing the canal a resulting rise in the audiometric curve of the tested ear was obtained, corresponding to the increase in bone conduction on the opposite (packed) side. This they believed could only take place if the ear phones were recording bone

19. Ersner, M. S., and Pressman, J. J.: Bone Conduction as an Element of Error in Audiometry, Ann. Otol., Rhin. & Laryng. **40**:448 (June) 1931.

conduction. The degree of hearing remaining in persons with destroyed labyrinths, which was shown with the use of the audiometer, was increased by prolonging the bone conduction of the well ear by packing the external meatus. This was taken as a further proof that bone conduction takes place from the ear phone and that the residual hearing in these deaf people as shown by the audiometer was actually bone conduction produced by transmission of sound through the skull to the opposite ear. Further observations were made by removing the ear phone an inch from the auricle. A marked diminution in the level of hearing in certain cases amounting to total cessation resulted.

In view of these findings, they concluded as follows:

(1) In Rinne negative cases, or in Rinne positive ears in which air conduction is less than bone conduction on the opposite side, the audiometric curve obtained with ear phones represents bone conduction and not air.

(2) This factor of bone conduction in the above mentioned type of case is sufficiently marked to give results which clinically are inaccurate and misleading.

(3) The audiometer, while most satisfactory in the majority of cases, is less so than tuning forks when air conduction is diminished below the bone conduction of one ear or the other.

I believe the point made by the essayists is well taken. When audiometers were first devised, telephone engineers claimed that with the telephone receiver all transmission of sound was by air conduction, the sound coming by way of the vibrations of the diaphragm. They emphatically denied that there was any conduction of sound to the skull bones by way of the vulcanized rubber portion of the receiver. The work of Ersner, Shambaugh and others seems to demonstrate definitely that there is a certain amount of conduction, apparently sufficient in certain types of hearing impairment, to give erroneous results with the audiometer. The audiometer is very useful in many ways; but it has certain defects that have been already pointed out by others than myself time and again.

In practice, for quantitative work, and especially for research problems, the audiometer is of immense value; but for the average otologist, the elicitation of a complete history, the proper use of a few good forks for the determination of the fundamental tests of hearing, the voice tests, the examination of the ears, nose and throat, and the careful correlation of all these facts, will enable him to make the correct diagnosis.

Becker [20] presented a method for exposing simulated deafness by using bone conduction tests. The principle involves the use of a stream of air against the tympanic membrane to exclude all sound reaching the cochlea by either air or bone conduction. A strongly vibrating

20. Becker, B. M.: Exposure of Simulated Deafness by Bone Conduction Tests, Laryngoscope **41**:653 (Sept.) 1931.

tuning fork, 128 or 256 double vibrations, is placed on the vertex or on either mastoid. The ear in question is tested first, the air being blown into it through a rubber tube placed in the external meatus and the fork placed over the mastoid on the same side. A person with true perceptive unilateral deafness will appreciate some sound, but may not be able to localize it. The malingerer, on the other hand, not realizing that the sound can be perceived in the other labyrinth, will deny hearing any sound. If he says he hears it on the opposite side, the tube is placed in the opposite normal ear and the same procedure repeated. The patient with true perceptive deafness will not hear anything. The malingerer will refer the sound to that ear even though he does not hear it there. Becker mentioned the fact that the Weber test has been used in cases of malingering, but only to give certain impressions and not for making a definite diagnosis.

Thus far I have not seen any test for simulated unilateral deafness which is better than the principle of the Stenger test and its modifications. The Stenger test has been discussed in detail in previous reviews in the ARCHIVES.[21]

Federici's[22] rather extensive article discusses the fact that the tuning fork placed on the tragus is heard longer than over the mastoid process except in cases of fixation of the footplate of the stapes in the typical form of otosclerosis (or as he more correctly said, otospongiosis). He described the results of tests in a number of different conditions with the following results:

When the external auditory canal is occluded by a plug of cerumen, the fork sounds over the tragus longer than over the mastoid; the same reaction is obtained in acute or chronic middle ear catarrh; also, but to a less degree, in cases of acute suppurative otitis media. In cases of chronic suppuration of the mastoid, hearing by way of the tragus is somewhat longer than over the mastoid but not in the same degree as in the normal cases. Where a radical operation has been performed on the mastoid with good surgical result, the test has proved negative, namely, the hearing by way of tragus was not longer than by way of the mastoid process. This result, it seems, vitiates the value of the tragus test so far as its value in diagnosing otosclerosis is concerned. In degeneration of the auditory nerve, the hearing by way of the tragus is longer than by way of the mastoid. In forty-eight cases of otosclerosis, the hearing by way of the tragus exceeded that

21. Sonnenschein, Robert: The Functional Examination of Hearing, Arch. Otolaryng. **1**:89 (Jan.) 1925; The Functional Examination of Hearing: Deaf-Mutism and the Education of the Deaf, ibid. **5**:48 (Jan.) 1927.

22. Federici, Frédéric: Nouvelle épreuve acoustique, Act oto-laryng. **16**:15, 1931.

by way of the mastoid by only a few seconds; in other words, the difference in hearing by way of the tragus compared with the mastoid is greatly diminished compared with normal cases or cases in which there is ordinary involvement of the middle ear or lesions of the inner ear. The author therefore believes that this is a very valuable sign in diagnosing fixation of the footplate of the stapes.

A somewhat unusual but rather significant statement is that made at the end of the article in small type, literally translated, that the author, at the moment of correcting the proof of his article, by chance saw a résumé of Lewis' original article in the *Annales des maladies de l'oreille, du larynx et des organes connexes* of July, 1925. The author therefore realized that in 1925, Lewis called attention to the fact that he had noted that a fork placed on the tragus is heard longer than over the mastoid or the root of the zygoma except in cases of fixation of the stapes. This occurrence again emphasizes the fact that many of the European otolaryngologists do not read the American or English literature. As a rule, their excuse is that they are too poor to subscribe for journals printed in the English language. Had this author at least have looked up the literature he would have found the abstract of the article in the French *Annales* of 1925, which was published several years prior to the time that he completed his paper. As discussing the idea of the so-called tragus test, permit me to quote from my review [23] of Lewis' original article.[24]

> Lewis believes that it is possible to differentiate between the ordinary conduction apparatus obstruction and stapedial fixation by a simple tuning fork test. By a stapes fixation he does not mean otosclerosis alone, but any condition which interferes with the proper mobility of the stapedial footplate.
>
> The vibrating tuning fork is held on the root of the zygoma, and is normally heard almost as long and as loud as over the mastoid antrum area. He states that it is normally heard longer on the tragus than on the zygoma or mastoid. The same relation exists when the external auditory canal and tympanic cavity are the site of a simple obstructive lesion. Cases of nerve deafness also show this same relation, that is, the fork is heard longer and louder on the tragus than over the zygoma or mastoid. This relation is reversed, however, in cases of known stapes fixation. Lewis uses a fork of between 96 and 256 double vibrations, or the audiometer with the bone conduction attachment. In making the Weber test, he uses four points on the skull, namely, the occiput, the vertex, the glabella and the symphysis of the jaw.
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> In performing the Weber and the Rinne tests, the author obturates routinely the external auditory canal during these tests. In this way he believes he obtains additional data concerning the presence or absence of obstruction of the tympanic

23. Sonnenschein, Robert: Functional Examination of Hearing: Review of Literature from November, 1924 to November, 1925, Arch. Otolaryng. **3**:60 (Jan.) 1926.

24. Lewis, E. R.: Laryngoscope **35**:109 (Feb.) 1925.

apparatus. A questionable lateralization by closing the canal on the same side may be unaffected or may be markedly pronounced.

When Lewis closes the external auditory meatus, as before described, he is really carrying out the Bing test, mention of which was made in Richard Lake's article in this review. In the paper by Minton and Sonnenschein on the use of a tuning fork stem for both air and bone conduction, read before the Chicago Laryngological Society, May, 1922, the words of Stefanini of Turin are quoted. This investigator found that when the tuning fork is placed on the tragus the conduction of sound into the ear is largely by air. I believe that there is every reason to assume, a priori, that the hearing by way of the tragus would be considerably longer than by way of the mastoid or zygoma; first of all, because air conduction normally is longer than bone conduction; and secondly, because by way of the tragus we have conduction not only by air but also somewhat by cartilage and bone because of the attachment of the tragus to the skull. In other words, it is simply another method of doing the Rinne test. Instead of having the conduction purely by air, which is usually done with the prong of the fork or by means of a tubing and stem as suggested by Minton and Sonnenschein, we have here the added factor of the air plus the bone and cartilage when the fork is placed on the tragus as originally shown by Stefanini.

I have often observed, and no doubt this has also been the experience of most other otologists, that in many cases of acute otitis media, in the first stages even, or at least after the condition has fully developed, there is a negative Rinne reaction, as the hearing by way of the mastoid is longer than by air or by the tragus. While Dr. Lewis uses a rather low pitched fork, I have always made the Rinne test with a fork somewhere between 256 and 500 d.v., preferably with the Bezold a-1 (435 d.v.) fork. The lower the pitch of the fork used, the more likely is the Rinne reaction to be negative. The higher the pitch of the fork, the more it tends to be positive. If the thesis of the author is correct, then it would seem that we must have a stapes fixation in many cases of acute otitis media, since the hearing of the forks by way of the mastoid or zygoma is often longer than by way of the tragus. This fixation evidently must be temporary, since we usually get the positive Rinne reaction again after the disappearance of all middle ear symptoms. How can it be shown definitely that we really have a fixation of the stapes footplate in all cases in which conduction of sound by mastoid or zygoma is longer than by the tragus?

Surely the history of the case, the sex, the effect of pregnancy, the triad of Bezold, the other tuning fork tests, etc., make the diagnosis of so-called otosclerosis fairly easy in most cases, without any reference to the tragus.

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Abstracts from Current Literature

Ear

INFLUENCE OF EPINEPHRINE AND OF EPHETONIN ON REFLEXES OF THE LABYRINTH. T. WASOWSKI, Monatschr. f. Ohrenh. **65**:967 (Aug.) 1931.

Wasowski points out that the most powerful vasomotor agent, namely, epinephrine, has found application in the treatment for diseases of the labyrinth, for it proved helpful in tinnitus aurium and in cerebral vertigo, not only when it was injected intravenously, but also when it was applied to the mucous membrane of the promontory in cases in which the tympanic membrane was absent. In investigations concerning the behavior of the caloric test following influence of epinephrine on the mucous membrane of the tympanic cavity, the author found that it intensifies the caloric reflex following cooling of the ear, and that it has an inhibitory influence when the ear is warmed. In order to determine the general influence of intravenous injections of epinephrine on the labyrinth, tests were made on rabbits. Attention was given not only to the variations in the ocular reflexes and to the caloric reflex of the labyrinth but also to the differences in the static and in the static-kinetic reactions. The eye reflexes were registered by means of a kymographion. In reporting the observations on the eye reflexes, the author states that in one instance following intravenous injection of 0.2 cc. of epinephrine he noted that the eyeball turned backward, made several horizontal fluctuations and remained in this position for two minutes. In another instance, in which 0.4 cc. of epinephrine was injected, a horizontal nystagmus was noted. A dose of 0.6 cc. of epinephrine resulted in a strong upward deviation of the eyeball. The author further reports his observations on the influence of injections of epinephrine on the caloric reflex of the labyrinth. He found that when the irrigation of the ear was done immediately following the intravenous injection, the reflex was retarded. When epinephrine was administered during the caloric nystagmus, the ocular movements were changed. Large doses of epinephrine may even cause cessation of the nystagmus. Studies on the behavior of the static and the static-kinetic reflexes following injection of epinephrine revealed that the injection of small doses of epinephrine does not cause changes in these reflexes. However, following injection of larger doses not only the progressive movement reactions but also the static reflexes are inhibited. The author further relates his experiences with ephetonin (hydrochloride of phenylmethylaminopropanol). A comparison of the action of the two substances reveals a certain similarity, only the action of ephetonin is somewhat weaker.

EDITOR'S ABSTRACT.

PROBLEMS OF EXPERIMENTAL SOUND INJURY BY WAY OF BONE CONDUCTION. H. BARTH, Ztschr. f. Hals-, Nasen- u. Ohrenh. **28**:397 (July 20) 1931.

Barth directs attention to investigations by Wittmaack which revealed that in animals changes can be produced in the cochlea by exposure to great noise. Wittmaack's observations were corroborated by some, but also caused considerable discussion, and the question whether the injurious action of sound takes place only by way of air conduction or also, perhaps exclusively, by way of bone conduction is still unsolved. The solution of this problem is of great practical significance in that it would help in adopting suitable prophylactic measures for workers who are obliged to work in noisy shops. After reviewing Wittmaack's experiments, the author reports his own investigations, on the basis of which he reaches the conclusion that Wittmaack's bone conduction theory of occupational

baryecoia cannot be proved, and that Siebenmann, who maintains that occupational baryecoia develops only by way of the influence of the noise on the middle ear, is right.

EDITOR'S ABSTRACT.

OTOSCLEROSIS EXAMINED BY MEANS OF POLARIZED LIGHT: PROBLEMS OF FIBRILS AND CEMENT LINES. M. WEBER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **28**:415 (July 20) 1931.

Weber shows that when the hematoxylin-eosin stain is used the fibrils of the bone tissues, which represent an essential structural element, do not receive sufficient consideration. However, if the decalcified section is examined by means of polarized light, the behavior of the fibrils can be observed. The two methods combined were employed in ten cases of otosclerosis and a number of new observations were made. First the normal labyrinthine capsule was examined in the polarized light and the existence of the fine-fibered, nonlamellar, so-called strandy marrow bone, which was first described by Meyer, could be corroborated. The author suggests the term strandy shell bone. Examination in the polarized light also revealed that on the margin of the otosclerotic foci, where in the hematoxylin-eosin preparation the blue stained humps protrude, and where the line of the cement substance is generally missing, the fibrils of the surrounding capsule end, as if they were cut off. The fibrils of the capsule often show concavities, into which fit exactly the humps that are visible in the hematoxylin-eosin preparation. Within the focus, the fibrils that appear "cut off" are attached to the long side of fibrils that have an entirely different structure, and they fill out the lacunae in the bone of the old capsule. The otosclerosis focus shows a framework of primarily coarse bundled fiber bones, and the wall casing of the cavities shows a fine fibered shell bone, which is partly free from lamella (strandy shell bone). Previous to the healing, longer strands do not develop, because the new formation is always interrupted again by disintegration. The otosclerotic focus represents a complete unit, which, in regard to its construction elements (cement substance, cells and fibrils), differs from the bone of the surrounding capsule. It is separated especially as far as the fibrils are concerned, and the only connection is probably by means of the marrow spaces. Otosclerosis may be considered as a new formation following a previous resorption. The author further describes the peculiarities in the cement lines, and he stresses that in order to solve the problem of the so-called blue mantles and of the sievelike perforations it is advisable to employ both methods, the hematoxylin-eosin staining and polarized light.

EDITOR'S ABSTRACT.

UNUSUAL SOURCE OF INFECTION OF THE PETROSA. C. VON EICKEN, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **21**:96 (June) 1931.

Von Eicken observed that pain in swallowing may be caused by an unusually long styloid process. In recent years he has therefore resorted to bimanual palpation of the tonsillar region and of the lateral pharyngeal wall whenever pains in swallowing could not otherwise be accounted for. In a considerable number of cases he found a styloid process of unusual length. He gives a detailed history of one case. Following resection of the lower portion of the styloid process an ascending periostitis developed, which led to sequestration. The inflammation spread to the base of the skull, where the outlet of the facialis is close to the styloid process. The facial canal and the adjacent cells of the mastoid process became involved, and gradually the mass of the mastoid process and the entire cell system became infected. Even the opening of the facial canal did not check the process; a labyrinthitis set in which, without impairing the surrounding bone, destroyed the function of the cochlea and of the semicircular canals. The author states that it is not at all rare for pathologic processes to spread from the mastoid and finally reach the pharyngeal wall, causing retropharyngeal abscesses; however, in the reported case, the infection took the reverse route. He reports that in the

other cases (nine in all) in which he resorted to resection of a portion of the styloid process, he did not observe such serious complications. He thinks that the infection may have been caused by the simultaneous removal of the chronically infected tonsil, although he had combined tonsillectomy in several other cases with the resection of a portion of the styloid process, without complications. Nevertheless, he thinks that it is advisable to perform the tonsillectomy first and wait until the tonsillectomy wound has completely healed before resection of the styloid process.

EDITOR'S ABSTRACT.

FIFTY CASES OF UNILATERAL DEAFNESS, SEVEN OF THEM CONGENITAL. A. MARSCHAK, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **21**:145 (June) 1931.

In 4,650 school children Marschak found 50 with unilateral deafness. Among these were 23 with an entirely normal tympanic membrane. In the other 27 the otoscopic findings indicated a tympanogenic deafness. For 16 of the first mentioned 23 patients the author assumes an acquired deafness, whereas in the other 7 the deafness was probably congenital. He considers congenital unilateral deafness as a special type of that developmental disturbance of the labyrinth which causes deaf-mutism. The anatomic changes are minute abnormalities in the structure of the cochlear apparatus, whereas the vestibular apparatus is generally normal.

EDITOR'S ABSTRACT.

PRESSURE ATROPHY OF THE BONY AUDITORY MEATUS. H. BEYER, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **21**:202 (June) 1931.

In discussing senile changes of the ear Beyer directs attention to the fact that besides the slitlike contractions resulting from the reduced expansion capacity of the walls and from the atony of the fibers that fasten the membranous portion to the temporal bone, there occur in old persons abnormally wide auditory canals, caused by pressure of plugs of cerumen or of epidermis, or resulting from senile atrophy. After reviewing the literature the author describes three cases from his own observation. In all three persons there existed a pressure atrophy of the external auditory meatus. In one of the cases the process had the aspects of otitis externa desquamativa, in which the glands, as the result of the cutaneous inflammation, were in a condition of constant irritation and had an excessive secretion. Inflammation and desquamation therefore spread from the wall of the auditory meatus directly to the epidermis of the tympanic membrane, which became irritated, and thus the hearing was impaired. Even the periosteum became involved; by the desquamation of the bone it gradually became eroded. In the two other cases, senility was the main factor, for here the pressure atrophy was without symptoms, the skin showing no signs of irritation. In spite of the fact that there were no other signs of erosion on the parietal bone, the forehead or the occiput, it must be assumed that the senile bone had only a slight resistance against the pressure from the ceruminal plug and thus became gradually atrophied. This also explains the absence of pathologic changes in the tympanic membrane and the relatively good hearing capacity.

EDITOR'S ABSTRACT.

ACUTE INFLAMMATION OF MIDDLE EAR DURING CHILDHOOD. M. MEYER, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **21**:217 (July) 1931.

Meyer reports the clinical observations on 200 nurslings and children with acute otitis, who came under his observation in the last two years. He discusses the local symptoms, the general reactions of the organism, the pathologico-anatomic findings, the various forms that otitis assumes and also the treatment. He found that the clinical aspects of acute otitis are much the same in nurslings, in older children and also in adults, and he considers it unnecessary to make differentiations merely on the basis of age. The age of the patient is only one of many

points that he thinks deserving of consideration. The difficulties in the estimation of otitis media in children, even when the technical difficulties in the examination are disregarded, are, on the whole, greater than in later life. This is mostly due to the frequency of intercurrent diseases, which mask the symptoms of the ear disease. Another peculiarity of otitis in children is that it is difficult to classify and for this reason each case demands individual observation.

EDITOR'S ABSTRACT.

NORMAL AND PATHOLOGIC EARS OF NURSLINGS AND CHILDREN. M. MEYER, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **21**:283 (July) 1931.

In the first part of the article Meyer discusses the symptomatology and the results of the microscopic investigations in cases of mucous-suppurative mastoiditis. Then he evaluates Preysing's theory that the hollow spaces, which develop in the middle ear of nurslings during otitis, represent a pneumatization process. Although most investigators have refuted Preysing's theory, the author concludes from his own observations that the hollow spaces are the product of an abnormal pneumatization process, and he agrees with Preysing, who designated them as "hollow space sprouts." In the third part of the article he discusses the transformation of the medullary spaces during pneumatization. In the summary he states that the extension of the embryonal connective tissue from the antrum into the medullary spaces can be explained without the actual ingrowing of the embryonal connective tissue, especially since this tissue is in a state of retrogression after birth. Even if it cannot be proved morphologically, it appears nevertheless probable that the connective tissue, which resembles the submucosa, becomes visible in the medullary spaces, because the cellular constituents of the lymph marrow retreat. The transformation into fiber marrow or into fat marrow then takes place much like in other bones, only for the local peculiarity that here, in the mastoid process, air spaces are formed by the epithelial activity.

EDITOR'S ABSTRACT.

SO-CALLED OTOGENIC, ACUTE, PROGRESSIVE OSTEOMYELITIS OF THE TEMPORAL BONE IN CHILDREN. W. BROCK, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **21**:295 (July) 1931.

Brock states that acute otogenic progressive osteomyelitis of the temporal lobe is extremely rare in children. He gives a short review of the literature and then describes a case that came under his observation. In order to gain a better understanding of the etiology and the pathogenesis of this disease, he made a careful postmortem examination, and the temporal bone was subjected to careful histologic examination. He also discusses the theories of pathogenesis that have been advanced by other authors and concludes that his theory of pathogenesis is still hypothetic in that he has no definite clinical and anatomic proofs. Since the cases are extremely rare, so that one otologist rarely has occasion to observe several, the author advises the exchange of observations on cases of this nature.

EDITOR'S ABSTRACT.

TESTING THE EQUILIBRIUM. A. BLUMENTHAL, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **21**:437 (Aug.) 1931.

After pointing out that testing the equilibrium is one of the most important diagnostic aids in the detection of nervous diseases and of the disorders of the labyrinth, Blumenthal calls attention to some of the shortcomings of the generally employed methods and goniometers, especially to those in which the inclined plane is used. He further describes a test method that he found valuable. Two scales are placed 1 cm. apart, the dials facing each other. On the standing platform of each of the scales an oblique line is made. These lines indicate the positions of the feet. If a person with one foot on each scale stands perfectly still and

upright, and if static disturbances are absent, the dials of both scales show the same weight. In patients with neurasthenia the dials generally indicate considerable fluctuations. Patients who show only slight staggering in Romberg's test show considerable fluctuations in the two scale test. The dial readings should be made two seconds after the patient has stepped on the scales, and then several readings should be taken, and the average should be taken as basis for the estimation. Only one dial has to be read, since the other one indicates the difference between the person's total weight and the weight indicated on the one dial. The behavior of the weight distribution in disturbances of the labyrinth is determined in that the normal labyrinths of test persons are stimulated either by rotatory experiments or by caloric stimulation with cool water (15 C.). If labyrinthine diseases such as fistulas have been cured, disturbances in the equilibrium do not persist; however, in labyrinthine defects that are caused by traumas, the compensation is not so rapid. The author describes the results that he obtained with the two scale test, but he also emphasizes that it should be considered not only that testing the equilibrium reveals the condition of the labyrinth, but that the equilibrium involves a number of static factors, of which one is the labyrinthine. Besides, by the latter, the equilibrium is influenced by symmetrical body structure, sensibility of the muscles, of the joints and of the skin, motoric muscle innervation and adequacy of the nervous centers. These different static components should always be considered, no matter what test method is employed.

EDITOR'S ABSTRACT.

Pharynx

ELECTROSURGERY AS AN AID IN RHINOPHARYNGOLOGY. LEE COHEN, Ann. Otol., Rhin. & Laryng. **40**:329 (June) 1931.

Inasmuch as electrosurgery has been accepted as an important acquisition by many who are working in special fields of surgery, the author feels that all rhinopharyngologists are compelled to recognize the need for this work and to make a thorough study of the principles involved in the use of the several different types of high frequency currents. He is convinced, after four years of experience, that there is a real place in the specialty for this valuable adjunct to surgery, but is unwilling to follow those enthusiasts who relegate long accepted surgical methods, such as that for the removal of tonsils, entirely to the past. Many conflicting views are still held as to the value of electrosurgery in complete removal of the tonsils. While the author is firm in his belief that it is the method of choice in selected cases, he strongly advocates clean surgical removal in healthy adults up to middle age, who present good surgical risks, and in all children in whom eradication of the tonsils is indicated. Electrocoagulation is especially valuable in aged persons, in the presence of tuberculosis, organic heart disease, nephritis or hypertension, and in any condition in which surgical methods are contraindicated. The absence of shock, the slight pain and reaction following its use and its economic advantages are factors highly in its favor. A plea is made for all those attempting this work to obtain a thorough knowledge of the physical effects of the various currents, as well as to be thoroughly trained in the anatomy and surgery of the field. The use of the method in the hands of the unskilled is highly condemned. A description of the technic as used by the author is given in detail.

SNAPP, Grand Rapids, Mich.

OCCURRENCE OF ACTINOMYCES-LIKE BODIES IN PALATINE TONSILS. J. HOFHAUSER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **129**:293 (Aug. 18) 1931.

Hofhauser states that in 1920, when he reported two cases of Actinomyces disease of the tonsils, he pointed out that besides the true actinomycetes there also exist micro-organisms that resemble actinomycetes. He then reviews the literature on the subject and finds that all authors agree that in histologic structure these organisms resemble actinomycetes. Some consider them true actinomycetes; others

think that they are incompletely developed actinomycetes, and still others consider them either saprophytes or a conglomeration of fungus threads and bacteria. This diversity of opinions induced the author to investigate the problem. Studies were made on the tonsils of fifty patients who had been operated on for chronic tonsillitis. Fungi were searched for in the matter pressed from the tonsils, and besides this serial sections were made from the tonsils. The results of these investigations are summed up as follows: 1. In the inoculated culture mediums there grew partly gram-negative and partly gram-positive bacilli and cocci, and in twelve instances there also were various fungi, most of them belonging to the genus Oidium. 2. In the inoculated white mice an induration developed, which usually became absorbed in the course of ten days. In several cases an abscess developed, but actinomycosis was observed in none of the animals. 3. The serial sections were stained with hematoxylin and eosin by the methods of van Gieson and Gram. Microscopic examination revealed various stages of inflammatory manifestations: The crypts were filled with inflammatory cells; in parts the lacunar pavement epithelium was preserved, while in other parts it was detached or infiltrated with inflammatory cells, mostly leukocytes. The lacunae were dilated in most cases and were usually filled with leukocytes and with desquamative epithelial cells. A true colony of actinomycetes could be found in no instance, but in fifteen of the fifty cases formations were found that in form and size resembled actinomycetes, although staining and consideration of the finer structure aided in the differentiation from true actinomycetes. For this reason the author is not in favor of the term Actinomyces-like bodies, but recommends the designation of corpora lacunaria. He concludes that true actinomycetes that later cause actinomycosis occur rarely in the tonsils, but that the corpora lacunaria are found comparatively often.

EDITOR'S ABSTRACT.

IRRITATIVE CONDITIONS OF CRYPTS OF PALATINE TONSILS (LATENT CRYPT TONSILLITIS). B. THIESBÜRGER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **28**:459 (Aug. 20) 1931.

Thiesbürger reports studies on the palatine tonsils. His observations are summed up as follows: 1. Systematic studies on sections of clinically a[illegible] anamnestically healthy tonsils of corpses revealed an unusually high percentage (71) of cases with simple inflammation of one or several crypts. 2. Even normally there are differences between the bacterial flora of the tonsillar crypts and of the surface of the tonsils, and this observation could be corroborated in studies on living persons. The conditions are similar to those in the case of appendicitis. The flora of the crypts is such, which even normally contains those micro-organisms that cause the clinically manifest forms of acute and chronic tonsillitis, and the clinically latent inflammations of the crypts. 3. In many cases of clinically latent inflammation of the crypts, a phagocytosis of certain micro-organisms by the leukocytes takes place simultaneously. This phagocytosis is the only sure sign of the pathogenicity of the micro-organisms in each respective case. Moreover, the bacterial phagocytosis is also a significant basis for the correct evaluation of the bacterioscopic and bacteriologic findings. 4. The author defines the clinically latent crypt tonsillitis. He maintains that not the macroscopic appearance of the tonsillar plugs, but rather the microscopic detection of more or less marked leukocyte admixture is decisive for the diagnosis of inflammation of the crypts. 5. Investigations with a combination of several methods justifies the assumption that diplostreptococci, especially hemolytic streptococci, are the main cause not only of the clinically latent crypt tonsillitis but also of the clinically manifest forms of acute and chronic tonsillitis. Judging by appearances, the specific tonsillitis, at least the Plaut Vincent type, begins with a transitory streptococcic tonsillitis. 6. The essential cause of tonsillitis is either an increase in the virulence of the bacteria or a decrease in the resistance of the organism. As a predispositional factor, the stagnation of the cryptic contents is of primary importance in the development of the inflammation of the crypts. As probable factors that are responsible for the prolongation of the process, the author considers horny plugs, cornification and perhaps also tonsil-

loliths, but he does not hold leptothrix nodules responsible. 8. It is very probable that the clinically manifest forms of tonsillitis begin with clinically latent, acute inflammation of the crypts.

EDITOR'S ABSTRACT.

CHRONIC TONSILLITIS AND CHRONIC TONSILLOPATHY. A. LINCK, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **21**:342 (July) 1931.

According to Linck, the term "chronic tonsillitis" is applied to a permanent pathologic condition of the palatine tonsils. It is an empiric term and it was selected because, on the basis of clinical observations, it was assumed that it was always due to chronic inflammatory changes of the palatine tonsils. However, careful investigations revealed that the pathologico-anatomic collective term "chronic tonsillitis" is not applicable to some tonsillar diseases, and therefore the term "chronic tonsillopathy was recommended. The term chronic tonsillopathy includes those conditions that are usually termed chronic tonsillitis but the two terms are not synonymous. Tonsillopathy is the more general term, including also those pathologic conditions of the tonsils in which inflammation is absent. In the main part of the article the author discusses first the pathology and then the clinical aspects of the various forms of chronic tonsillopathy, and he discusses particularly the therapeutic and prophylactic significance of tonsillectomy.

EDITOR'S ABSTRACT.

RECENT PROGRESS IN THE SPHERE OF ACUTE DISEASES OF THE THROAT (AGRANULOCYTOSIS AND LYMPHOID CELLULAR ANGINA). W. SCHULTZ, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **21**:367 (July) 1931.

In the introduction, Schultz defines the terms "agranulocytosis" and "lymphoid cellular angina." He points out that both terms are derived from hematology. The granulocytes, the absence of which is the essential factor of agranulocytosis, are polymorphonuclear neutrophilic and eosinophilic leukocytes. In hematology, "lymphoid" does not signify resembling lymphocyte, but lymphoid leukocytes are basophil nongranulated cell elements. The author further describes the symptomatology, the characteristic changes in the blood and the treatment for agranulocytosis. In regard to lymphoid cellular angina, he states that it is now considered as a generalized systemic disease of the lymphatic organs. In the first cases observed monocytic blood elements predominated, and the disease was designated as monocytic angina. Later observation revealed, however, that lymphocytes predominated, and for this reason it was termed lymphoid cellular angina, the term lymphoid including both lymphocytes and monocytes. However, the author takes exception to the recently advanced theory that lymphoid cellular angina is identical with Pfeiffer's glandular fever. Pfeiffer's glandular fever is usually of short duration, affects mostly children and is easily communicable, whereas lymphoid cellular angina lasts longer and affects mostly young people between the ages of 15 and 25, but not children; in fifty cases of lymphoid cellular angina there never was a direct transmission. The author also directs attention to certain peculiarities in the symptomatology of lymphoid cellular angina, and then he discusses the differential diagnosis. The prognosis he considers as comparatively favorable, and the treatment as mostly symptomatic.

EDITOR'S ABSTRACT.

Larynx

USE OF NEW ARTIFICIAL PALATE IN PALATOGRAPHIC TESTS ON STAMMERERS. F. GUMPERTZ, Monatschr. f. Ohrenh. **65**:1095 (Sept.) 1931.

By using celluloid palates Gumpertz was able to make palatograms that resemble those of the natural palate. Thus he was in a position to compare palatograms of the same sounds produced by the same person at different times, and he could also study the modification of the formation of certain sounds by

neighboring sounds. The use of this method on stammering children revealed the following: The sites of articulation and the positions of the tongue for the various sounds are less constant in the stammerer than in the normally talking person. The stammerer soon shows articulatory fatigue. Whereas the pressure of the tongue in the normal adult is symmetrical on both halves of the palate, the unsteadiness in the sound formation by the stammerer is frequently characterized by a greater pressure of one side of the tongue and by wider apposition of the tongue to the palate. Acoustically this asymmetry is not expressed; especially in the s sound it does not necessarily have to be heard as a lateral sigmatism. The palatogram shows still other anomalies in the stammerer that are not perceptible with the ear: for instance, in one case, a tendency to planelike apposition of the tongue in enunciation of t and in another case an s-like position of the tongue in enunciating t. In cases of so-called kappacism one hears instead of k, a t or a t-like sound; yet the palatogram already shows the lingual positions of articulation characteristic for k. These occur either alone or with simultaneous articulation of t. The intermediate sound between k and t can have just as well a t articulation on the soft palate, as a k articulation on the anterior portion of the hard palate. The kappacism of the stammerer resembles in regard to its articulation the intermediate sounds of certain dialects. The transition from t to k is changeable in regard to sound value as well as palatographically. The fact of k articulation in apparent speaking of t is, just as the medially closed n-line in stammerers with medically gaping t-palatogram, a proof that stammering a sound is not merely a mechanical incapacity but is a speech habit. That an intended t in an isolated syllable is formed atypically, but in a polysyllabic sound combination or as substitute for an intended k is formed typically, likewise is a manifestation of a speech habit. In the stammerer the articulation of the consonants is influenced not only by the articulation of the adjoining vowel (contact assimilation), but by the consonant at the beginning of the following syllable (distant assimilation). The t-palatogram may be influenced by a following p; the k-palatogram may become practically identical with the t-palatogram if a t follows. However, a modification of the articulation of k by a following p or f is not demonstrable. A preceding n in the combination nk does not tend to palatalization, i. e., to forward shifting and amplification of t characteristics; even if palatalization does exist in the nk combination, the k characteristics are often more pronounced than in an isolated k. In stammering that is due to organic factors the assimilation tendency, as observed in the developmental stammerer, is absent. When nd and nt were intended in a case of organic stammering, ng and nk were heard, but the palatogram revealed the line corresponding to the dental n, whereas this line was absent when the posterior n was normally formed i. e., when ng and nk were intended. In the conclusion the author expresses the opinion that contact assimilation in the organic as well as in the developmental stammerer is not merely the result of simplification of movements in accordance with the mechanical principle of least exertion, but that it is primarily psychologic.

EDITOR'S Abstract.

Nose

PATHOGENESIS OF ATROPHIC RHINITIS. W. L. SZÁLKA, Monatschr. f. Ohrenh. **65**:848 (July) 1931.

In the introduction to his paper, Szálka states that the causes of atrophic rhinitis, also termed ozena, are still not completely understood. He differentiates between genuine and secondary atrophic rhinitis and then reviews and evaluates the various theories of pathogenesis. He concludes that ozena is caused and maintained by an inflammation, which is chronic and of an alterative character. The chronic inflammation may be caused by the abnormal width of the nose and by hypovitaminosis. This theory is corroborated by clinical observations and by the results of therapeutic measures that had been based on it.

EDITOR'S ABSTRACT.

PRIMARY STAGE OF SO-CALLED TRUE OZENA. J. BIRKHOLZ, Monatschr. f. Ohrenh. **65**:857 (July) 1931.

Birkholz gives the clinical histories of three children with ozena, which he considers of special interest, because they disprove the theory that the atrophic process in ozena is preceded by a hyperplastic process, an assumption which for the last fifty years had been accepted by many investigators. In all of these cases the clinical histories previous to the development of ozena were available, and a hypertrophy of the mucous membrane was mentioned in none of them. The ozena developed directly. All at once, it was there. The author thinks that true ozena is neither of purely endogenous origin, nor is it caused by exogenous factors alone. Moreover, it should not be considered as a local disorder, but the patient's general condition should always be taken into consideration.

EDITOR'S ABSTRACT.

DISEASES OF ACCESSORY CAVITIES IN HUMAN BEINGS AND DOGS AS OBSERVED ON DISSECTION MATERIAL. O. URBACH, Monatschr. f. Ohrenh. **65**:920 (Aug.) 1931.

On the basis of his observations, Urbach arrives at the following conclusions: 1. In dogs diseases of the accessory cavities are extremely rare. 2. In human beings the antrum of Highmore is the most frequently affected (92 per cent) of the accessory cavities. 3. The recent increased incidence of disturbances of the accessory cavities in comparison to former reports, the author considers due to the influenza epidemic. 4. The disease of the antrum of Highmore is always bilateral. 5. Only in 16 per cent of the patients with disorders of the accessory cavities the antrums of Highmore alone are affected; in all other cases some of the other accessory cavities are involved. The ethmoid sinus is involved in 56 per cent of the cases, the sphenoid sinus in 44 per cent and the frontal sinus in 20 per cent. 6. On the basis of the clinical symptoms only acute and chronic highmoritis can be differentiated. Other forms such as granulous, polypous or fibrous can be recognized only during the operation. 7. Puncture is of no value for the diagnosis and may even be harmful. 8. Operative treatment of the antrums of Highmore should always be done first, and only in the event that this operation is not effective, and if special indications exist, surgical interventions on the other cavities should be resorted to.

EDITOR'S ABSTRACT.

NASAL FRACTURES IN THE ROENTGEN PICTURE. H. LÖLLKE, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **21**:80 (June) 1931.

Löllke states that although larger fractures of the bony frame of the nose are generally readily recognized, the smaller fractures cannot always be diagnosed merely on the basis of the clinical signs, for the classic fracture symptoms, such as abnormal movability and bony crepitus, are not present in small fractures, and the pain is so generalized that it likewise does not permit localization of the fracture. For this reason roentgenoscopy is advisable. In reviewing the literature on roentgen examination of nasal fractures, the author evaluates the various methods of exposure, namely, the lateral, occipitofrontal, endonasal and endoral forms, and roentgenoscopy of the hanging head, in which the film is laid on the outside of the nose and the central ray passes through the floor of the mouth. The most suitable form is the lateral, for in this exposure the nasal bones are not overshadowed by other bones of the cranium. The method employed at the Berlin university clinic is as follows: The 9 by 12 cm. films are held against the cheek and temple, parallel to the sagittal plane. The tube is at a distance of 6 cm.; the duration of the exposure is one second, the tension 40 kilovolts and the current strength 90 ma. The central ray passes through the middle of the bony nasal frame. The use of smaller films (tooth films) is in most instances not advisable, for although they produce sharp pictures, they show too small a portion; in some cases, however, they are a great help. The author further discusses the details

of the nasal roentgen picture. He shows how the lines produced by the sutures can be differentiated from the lines produced by fractures, and he also states where the fractures are generally to be found. In the last part of the article he discusses the healing process in nasal fractures. On the basis of observation and animal experiments he assumes that the healing process of the fractured nasal bones takes place not by callus formation but rather by connective tissue coalescence.

EDITOR'S ABSTRACT.

NONFEBRILE SINUS THROMBOSIS WITH PARALYSIS OF THE ABDUCENS. H. BUSCH, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **21**:165 (June) 1931.

In the discussion with which Busch begins his paper, he points out that the reported case is noteworthy for two reasons: (1) There was no fever and hardly any symptoms, and (2) the abducens became involved. That changes on the sinus transversus develop entirely without, or with only slight, symptoms is a rare but not an unknown occurrence. Fever was absent in the reported case, probably because the softened and suppurated thrombus in the middle of the sinus obstructed the venous channel so that pathogenic organisms could not enter the circulation, and also because the suppurated thrombus had perforated the wall of the sinus and thus had unburdened it. In regard to the second unusual aspect, namely, the paralysis of the abducens nerve, the author, after discussing various possibilities of pathogenesis, comes to the conclusion that in this case the sinus thrombosis was the only (mechanical) cause of the involvement of the abducens nerve, for following the operative removal of the thrombus the paralysis disappeared. In the last part of the article the diagnosis, prognosis and therapy of such disorders are discussed.

EDITOR'S ABSTRACT.

Miscellaneous

PRIMARY STAPHYLOCOCCUS INFECTIONS OF THE NOSE, LIPS AND FACE. IRWIN I. KOSLIN, Ann. Surg. **94**:7 (July) 1931.

Of eighteen patients with primary staphylococcus infections of the nose, lips and face admitted to the wards of the Lebanon Hospital, six died; four of them were men and two, women, ranging from 24 to 50 years of age. In all the fatal cases, blood cultures were positive for Staphylococcus aureus. The cavernous sinuses were thrombosed on one or both sides in four cases; meningitis occurred in one case, and septicemia in only two. In all cases, the condition began with minor trauma and the spontaneous appearance of a "pimple," which subsided, was disturbed by trauma or continued with increasing severity. From a review of the anatomy of the parts, the author finds that the thinness of the skin, with its rich vascular system and its close relationship with the dependent important venous and lymphatic structures, favors extension of the infectious process and brings it closer to the cavernous sinus. Involvement of the facial vein, because of its patency and the absence of valves, allows the infection to spread in either direction. There is no demonstrable direct connection between the lymphatics of the face and those of the skull. The author is conservative in his attitude toward treatment, favoring rest in bed and warm wet dressings; ligation of the facial vein and local surgical intervention are not favored.

L. M. WIEDER, Milwaukee.

HEADACHE AND OTHER NERVOUS DISORDERS IN THEIR RELATION TO THE SPHENOPALATINE GANGLION. E. V. ULLMANN, Monatschr. f. Ohrenh. **65**: 829 (July) 1931.

Ullmann shows that headaches which are not due to suppurative processes or to tumors, also neuralgias and other painful nervous manifestations in the region of the head, can be combined into one syndrome, or belong to the same group of

symptoms. This disorder, or better, this group of symptoms, had not received sufficient consideration until Sluder explained them. Not only headaches and neuralgias but also manifestations, which are usually combined under the name of functional or hysterical disturbances such as: globus hystericus, singultus, glossodynia, gnashing of the teeth, and myalgias in the region of the throat and neck, can be counteracted either by anesthetization or, or by injection into, the region of the sphenopalatine ganglion. The author gives a detailed description of the treatment and stresses that anesthetization should always be tried first in order to determine whether injection treatment is advisable. The injection is done with 0.5 cc. of 86 per cent alcohol to which phenol has been added (5 parts of phenol to 95 parts of alcohol). The technic of the treatment is comparatively easy and can be done by any experienced rhinologist, and since it does not involve dangers it should be resorted to whenever it seems advisable.

EDITOR'S ABSTRACT.

SIMPLE HYPOPHYSEAL OPERATION. J. TREER, Monatschr. f. Ohrenh. **65**:841 (July) 1931.

After classifying the various types of hypophyseal tumors, Treer evaluates the twenty-two surgical methods that have been recommended for the removal of hypophyseal tumors. He first discusses the older intracranial methods and then the extracranial methods. The majority of the surgeons who employ the latter approach the operative field through the septum. Several of the transseptal methods have the advantage of the submucous removal of the septum. This is helpful because: (1) the operative region itself is free from blood; (2) the sphenoid sinus is reached by a sterile route; (3) the sphenoid sinus can be opened more easily after the mucous membrane and the periosteum have been removed from its anterior and inferior wall; (4) after the sphenoid sinus is opened, orientation is not impaired by bloody portions of the mucous membrane. With these advantages in view, the author devised a new surgical method, which combines the advantages of several other methods. He gives a detailed description of the various stages of this method.

EDITOR'S ABSTRACT.

PATHOLOGY OF OTOGENIC FACIAL PARALYSIS. FREMEL, Monatschr. f. Ohrenh. **65**:950 (Aug.) 1931.

After reviewing some of the literature on otogenic facial paralysis, Fremel states that the pathology of facial paralysis is still largely hypothetic. The rare opportunity to study a case of facial paralysis in histologic sections induced the author to review the 104 cases of facial paralysis that were observed in his clinic during the last eight years. First he discusses 10 cases in which facial paralysis developed during acute otitis. These patients underwent antrotomy, but although the surgical reports mention perifacial cells and perifacial osteomalacia, nothing is said about the appearance of the nerve itself. In all these cases the facial paralysis disappeared again, and from this it is concluded that in cases of this nature there is probably a mechanical etiologic factor such as pressure. The author further discusses operative paralysis. In some of the patients the paralysis was noted immediately after the anesthesia had terminated, and in others it developed hours and even days after the operation. The majority of cases of facial paralysis developed in the course of chronic suppurations of the middle ear and in most of these patients the labyrinth was involved. The author further states that his material does not corroborate the assertion of other observers that facial paralysis is more frequent in children than in adults. Among his patients were 95 adults and only 9 children. In the last part of the article the histologic findings on the petrosa of a man, aged 62, are described. Facial paralysis developed in the course of an acute otitis, and the patient died soon after the surgical intervention. The petrosa was prepared in the usual manner. Reproductions of three sections are given. The first section shows the destructive process in the nerve.

The suppurative infiltration of the mucous membrane of the middle ear perforated through a fissure in the bone into the canal and destroyed the nerve by abscess formation. At this site the canal contains only an abscess cavity. Above and below the nerve abscess the infiltration continues in the nerve. Section two shows the nerve distal from the abscess. The canal is empty; that is, the nerve has degenerated and so also has the little branch going to the musculus pyramidalis. The surrounding bones show a severe otitic process, and it is probable that the paralysis is a partial manifestation of this process. A third section shows the facial canal medial from the focus of destruction. Here the nerve is mostly intact but shows here and there degenerated fibers and numerous osteoclasts on the wall of the canal with interstitial infiltration of the nerve. In this case the pathologico-anatomic form of nerve injury is a suppurative inflammation, originating in the inflammation of the middle ear and of the bone, and can therefore be considered as a partial manifestation of the process of the middle ear. However, this case does not justify generalizations in regard to the pathology of other cases.

EDITOR'S ABSTRACT.

PERCEPTION OF DIRECTION OF SOUND. A. REJTÖ, Monatschr. f. Ohrenh. **65**:959 (Aug.) 1931.

In the introduction to his paper, Rejtö compares binaural audition with binocular vision, and "plastic" hearing with stereoscopic vision. He points out that in human beings the orientation by means of hearing is not developed to the same degree as is the stereoscopic vision. Whereas we can yet judge the depth of objects with considerable exactitude at a distance of 10 meters, we cannot determine exactly the distance of the sound source, but recognize only the direction from which the sound comes. In order to gain a better understanding of direction audition, the author made tests on 100 persons, some of whom had normal hearing capacity and in some of whom it was impaired. It was found that as soon as the examined persons were permitted to move the head, the detection of the direction became more correct. Correct vision also aids in determining the direction of a sound source, provided the person is permitted to move the head. Persons in whom the hearing capacity of one ear is impaired frequently confuse the direction of a sound by projecting the sound source from the direction of the deaf ear into that of the normal ear. From this the author concludes that the basis of the detection of the direction of a sound source is the hearing with two ears. However, the incomplete perception of sound direction can be improved by auxilliary apparatus, especially by the eyes. The change of position of the organ of reception, which is effected by movements of the head, is also of great significance. The movements of the head and the reflexes of the eye muscles that are necessary for detection of sound direction are induced by labyrinthine stimulation. But while formerly this action of the labyrinth was considered as resulting only from central reflexes of the auditory nerve, it has more recently been demonstrated that the sound waves stimulate the static apparatus also directly. In the summary, the author stresses once more that binaural audition is the essential factor in determining sound direction, but changes in the position of the head, that is of the ears, and movements of the ocular muscles are also significant.

EDITOR'S ABSTRACT.

CONSTITUTION AND SPEECH DISTURBANCES. T. S. FLATAU, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **21**:63 (June) 1931.

Flatau points out that in a paper on the treatment for stuttering, which he read before the German society for voice and speech correction in 1928, he called attention to the fact that Jaensch's researches on the characteristic features of the physiology and pathology of the psychophysical personality, and especially the studies on capillaroscopy, have shown a way that will lead phoniatrics out of the stage of hypothesis toward exact comprehension of its psychophysical correlation.

It is observed that children with speech disturbances often have one or several other defects, such as premature or retarded sexual development, infantile myxedema, an asthenic and eunuchoid habitus, hermaphrodism, dysthyreosis and other disturbances of the hormonal equilibrium, motor infantilism, hereditary syphilis, congenital debility, obstetric trauma, spasmophilia, paralysis, hydrocephalus, microcephalus, epilepsy, disturbances in affect, inhibitions, depressions, anxiety, phobias, hypersensitivity, fitfulness, excessive timidity and anomalies of walking writing and facial expression. This concurrence of speech disturbances with other anomalies indicates the constitutional character. The author defines constitution and further differentiates between two main groups of persons with speech defects, the neuropathic and the psychopathic; he then reports results of studies on patients with speech disturbances. The patients were subjected to cranial measurements, roentgenoscopy of the head, especially of the sella, electrical irritability tests, psychotechnic and psychologic examinations and capillaroscopy. The results of the capillaroscopy are described in detail, and several case reports are given. The author does not wish to overemphasize the significance of capillaroscopy; however, he found that it permits a clear diagnosis and careful observation during the course of treatment. He states that in most cases a combined phoniatric and lipoid therapy proved helpful. The latter was employed in the form of a lipoid iodine preparation. In cases of hypothyreosis or hypophyseal disorders the respective organ preparations were helpful, and calcium, iron, bromine, phenobarbital and vitamin preparations were likewise given if necessary. The phoniatric treatment consisted of proper speech exercises.

EDITOR'S ABSTRACT.

DIVERTICULA OF THE HYPOPHARYNX. G. LANGE, Ztschr. f. Laryng., Rhin. (Teil 1: Folia oto-laryng.) **21**:375 (July) 1931.

After a general discussion of the symptomatology, the diagnosis and the methods of examination, Lange gives the clinical history of a man with a diverticulum of the hypopharynx. In regard to the surgical treatment, he says that formerly it was considered extremely hazardous, because of the dangers of phlegmous, of mediastinitis and of aspiration pneumonia. However, recent improvements in surgical technic, and especially the introduction of local anesthesia, have eliminated most of these complications. The author further describes and evaluates the various operative methods, especially the one that was successfully employed in the reported case.

EDITOR'S ABSTRACT.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

HOWARD C. BALLENGER, M.D., *President*

Regular Meeting, Oct. 5, 1931

THE MECHANICS OF AUDITION. DR. A. G. POHLMAN.

Audition is dependent on the conduction of vibrations that lie within the audible range to the end-organ, and on the transformation of the stimuli at the end-organ into action currents which are translated by the brain into terms of the sources. It is well known that the development of the vertebrate ear seemingly violates the physical requirements essential to an organ of great sensitivity, because the perceptive apparatus is an immersion end-organ, and because air sounds are almost completely reflected back in their conduction to water. The development of an immersion end-organ, however, fulfils the physiologic requirement of placing the perceptive apparatus under conditions of acoustic insulation, which permits great sensitivity in response. The acoustic insulation is overcome through the development of a middle ear that acts as a transformer to match the differences in resistance in the two mediums. The evidence seems to show that the middle ear apparatus is more efficient in the conduction of sounds of weak intensity, and that as the loudness of the signal rises, the intensity of reaction in the internal ear becomes proportionately less. This type of transformer is accordingly one of limited intensity reaction and affords protection to the auditory end-organ by acting as a shock absorber on intense sounds. The experimental evidence also indicates that the middle ear apparatus is about equally efficient in the conduction of air sounds of all frequencies to the internal ear as opposed to the generally accepted idea that the conduction through the ossicles is limited to low frequencies. The problem of the mechanics of the middle ear has been obscured through the attempts to interpret unreal phenomena in audition, such as paracousis willisii and prolonged bone conduction. Prolonged bone conduction is a real phenomenon only when the perceptive apparatus is normal, the sound conduction system functional and the drum membrane loaded or the external canal occluded. Under other conditions, sounds conducted by bone are conducted directly through the wall of the otic capsule and irrespective of the mechanical conditions of the middle ear. This interpretation establishes the value in differential diagnosis of the comparison between acuity of hearing by air conduction and that by bone conduction.

I am opposed to the usual interpretation of conduction deafness and believe that the accepted explanation is dependent on the degree of presbyacousia in the examiner, on the inadequacy of the test methods before the invention of the audiometer and on the sensitivity of the internal ear as a function of the frequency. The experimental evidence also opposes the accepted idea that the ossicular chain acts as a lever which decreases the amplitude and increases the force with which the vibrations of the drum membrane are propagated to the labyrinth. Comparative anatomy suggests that a leverage action is present in all forms of animals with middle ear apparatus, and that this leverage acts as shock absorber against a displacement due to topographic changes in the region. There is no evidence that the intrinsic muscles act as an accommodative system, and while the stapedius muscle in man has been observed to react to sound stimulation, this is not the case in the tensor tympani muscle. The middle ear apparatus may accordingly be interpreted as one with efficient conduction of audiovibrations of

all frequencies and most efficient for those of low intensities. It may also be regarded as a shock-absorbing mechanism.

The morphology of the internal ear has been generally interpreted in terms of the mechanical analysis of pitch and predicated on the assumptions originally made by E. H. Weber that the otic capsule is inelastic, the liquid is incompressible, and the round window is the only compensation opening that permits displacement of the stapes. It is on these three premises that all theories of analysis of pitch that call for transverse vibrational responses in the cochlear duct and its contents have been based. The three premises, however, are not facts, and the evidence seems to substantiate the idea that the morphology of the internal ear has been misconceived. The experimental findings indicate that the round window is not related to the function of sound analysis.

The explanation as proposed seeks to eliminate the theories of indirect activation and to substitute one of direct activation in which the auditory cells themselves respond directly to the vibrations passing on or through them. This explanation suggests that the serial arrangement in the end-organ is dependent on the development, which also accounts for the dimensional differences in the cochlear duct and its contents. The experimental evidence favors the idea that the morphology may be interpreted as a protective mechanism against transverse vibrational displacement in the internal ear.

DISCUSSION

DR. GEORGE E. SHAMBAUGH: The problems of hearing are intriguing, chiefly because the reactions are physical and seem, therefore, more capable of analysis than are those of perception of light, for example, for one grasps the physics of hearing more readily than the chemistry of seeing.

One hardly knows just how to begin a discussion of Dr. Pohlman's paper; also one probably will have difficulty in knowing just where to stop. According to Dr. Pohlman's criticisms, there is little basis for any theory of hearing, but, as a matter of fact, people do hear in spite of these criticisms, and they hear with the organ of hearing.

Two factors are necessary in the physiology of hearing. The first is the existence of a mechanism for collecting sound waves that originate in the outer air and conducting them into the head, where they come in contact with the endings of the eighth nerve. The second is a mechanism for transforming these physical sound impulses into nerve impulses, and the transmission of these nerve impulses to the centers in the cortex where they are interpreted as sound.

We are satisfied about the function of part of this so-called sound-conducting mechanism. The auricle, for example, is the mechanism for collecting the sound waves and directing them into the external meatus, but it is of relatively little use in man. It seems to be much more important in many lower animals. Again, the external meatus is the means for getting the sound waves into the head. It is not such an important mechanism, for if you put your fingers in your ears and block off the external canal, every one in this room will hear what I am saying without my raising my voice. In the same way, a child born with atresia of both external auditory canals experiences little difficulty in hearing, provided the developmental anomaly does not involve the inner ear also. The ordinary voice is heard easily at the length of a large room by such a child.

It should be clear that operations to open the external auditory canal in order to allow sound waves to reach the labyrinth are not necessary, because if the labyrinth is involved such an operation is of no use and if the labyrinth is not involved the operation is not necessary.

The next step is a more difficult one, namely, the transference of the air-borne sound impulses into the fluids of the labyrinth. It is generally believed that the drum membrane and the chain of ossicles serve as a series of levers for increasing the intensity of the sound impulses which impinge on the drum membrane and finally reach the fluids of the labyrinth by way of the footplate of the stapes. Some

idea of the increase in intensity that is brought about by this chain of ossicles can be visualized by comparing the size of the drum membrane with that of the footplate of the stapes.

The clinician becomes rather skeptical as to the value of this leverage mechanism. Any one who has had much of a background of clinical experience has encountered this situation: the drum membrane gone, the chain of ossicles exfoliated and the stapes intact in cases in which the defect in hearing is so slight that it is scarcely appreciated by the patient. A careful test will reveal some defect in hearing, but it may be very slight; this mechanism for transferring the air-borne impulses to the fluids of the labyrinth cannot, therefore, be of great importance.

The important problem is the question of how the physical sound impulses get into the labyrinth. Is it through the chain of ossicles by way of the footplate of the stapes in the oval window, or is there another route by way of the round window and the membrana tympani secondaria? The practicing otologist has before him a mass of evidence on the interference with sound waves reaching the labyrinth when there is anything impairing the mobility of the stapes. Fixation of the stapes does interfere with the transferring of the sound waves to the labyrinth, and it interferes more with the transference of low pitched notes than with that of high pitched notes. The high pitched notes are somewhat interfered with, but by no means to the same degree as are the low pitched notes. This is an everyday experience that the practicing otologist encounters.

The question comes up here whether the high pitched notes may not be conducted into the labyrinth by some other route than by the chain of ossicles, and the suggestion has been made that the low pitched notes reach the labyrinth by means of the conducting mechanism and the oval window, whereas the high notes reach the labyrinth by the route of the round window. But there is an objection to this hypothesis: There must be some place in the tone scale where low pitched notes and high pitched notes are conducted equally well by the oval window and by the round window. This would create an impasse, which, as a matter of fact, we do not experience in our hearing for any part of the tone range.

There is a very simple explanation of why the low pitched notes have more difficulty in being transmitted by a stapes the movements of which have been impaired than have the high pitched notes, and it is simply that the vibrations of the stapes produced by the low pitched note are of greater excursion than are those of the high pitched note. The higher one goes in the tone scale the less these excursions become, and even complete bony fixation of the stapes does not seem to interfere with the passage of the high pitched note directly through solid bone to the fluids in the labyrinth.

There was an interesting experiment carried on by Siebenmann which threw a sidelight on this problem of sound conduction. We are all familiar with the results produced by overstimulation by high pitched whistles in a guinea-pig confined in a box. These degenerations of Corti's organ were not produced when the ossicular chain was broken by the resection of the long process of the incus, for example.

The mechanism for transforming the physical impulses of sound into nerve impulses is, as I have said, a necessary part of the function of hearing. There are two approaches to the solution of this problem. One is to study the phenomena of hearing, as, for example, tone analysis, and then try to visualize some sort of mechanism in the internal ear that can function so as to account for peripheral tone analysis. The physical resonators constitute the mechanism that seems most suitable for performing this function. With a physical resonator in mind, one examines the cochlea to see what structure is there that can perform such a rôle. This is the method that has usually been adopted. It was the plan followed by Helmholtz. He was impressed by the phenomenon of physical resonators as a means for tone analysis, and inquired of anatomists whether there was a structure in Corti's organ that could fill this rôle. He selected the rods of Corti as the rod resonator; as these vary in size from one end of the cochlea to the other, he felt that they might act in this manner, responding in different areas to tones

of different pitch and, by jarring the neighboring hair cells, bringing about their stimulation. Just how this stimulation of the hair cells by jarring was accomplished was not elucidated. He gave up this idea of the rod resonators, because birds and alligators, for example, did not have rods but, nevertheless, could hear well, and he selected the basilar membrane, the radiating fibers of which vary in length from one end of the cochlea to the other, as a physical resonator, because he thought it might possess the physical properties of a string resonator. As a matter of fact, a careful study of this membrane reveals definite objections to its acting in this manner. For example, as one approaches the lower end of the basal coil, the membrane loses entirely its resemblance to a string resonator and becomes a thick, wedge-shaped structure, quite incapable of filling the rôle assigned to it. The fact that it is a vascular structure shows that it is not capable of vibrating at all times in the same manner to the same impulses.

There is a more fundamental way of approaching the problem of deciding how the transference of physical impulses to nerve impulses takes place, namely, to study the structure of the organ itself and see whether a conclusion can be reached as to what the particular function of the various parts of Corti's organ may be, and then to build up a theory that will best explain the phenomena of hearing. This reasoning is very simple. The hair cells are undoubtedly the mechanism in which the transference from physical to nerve impulse takes place. The stimulation of the hair cells is unquestionably accomplished by irritation applied to the projecting hairs. This irritation cannot reach the hairs by impulses passing through the endolymph, because the hairs do not stick up freely into this fluid but are attached to the under-surface of the membrana tectoria. I was the first to demonstrate this important anatomic relationship, in 1907. Others in this country soon verified this finding, and more recently Held and Wittmaack have both observed the same condition.

It is clear, therefore, that stimulation of the hair cells must be accomplished by some interaction between the membrana tectoria and the projecting hairs. There are two ways by which this interaction may be brought about. One is by movements of the membrana tectoria itself and the other by movements of the membrana basilaris. From a biologic standpoint, the membrana tectoria is the logical structure for responding to impulses in the endolymph. In the first place, this brings the reactions of the cochlea into accord with the reactions in the two other types of end-organs in the labyrinth, the macula and crista acustica, in which the superimposed structures, the otolith membrane and the cupola, are the structures that move to stimulate the hair cells. In the second place, sound impulses reaching the labyrinth via the oval window impinge first and most forcibly on the membrana tectoria. The membrana vestibularis (Reissneri) is such a delicate, filmlike structure that it offers no interference to impulses passing directly through the scala vestibuli to the scala media.

The question arises as to what is the manner of response in the membrana tectoria. This structure varies in size from one end of the cochlea to the other, and its size must influence the physical response to impulses passing through the endolymph. To say that it can or cannot react in a particular way is unwarranted, since we know so little about the structure of the membrana tectoria. It is an elusive membrane. My feeling is that we should accept that kind of response in the membrane that will best explain the phenomena of hearing, and that the efforts to fix on the membrana basilaris a function that biologically it is not intended to perform are misdirected.

Models have been constructed to show how the structures may respond to sound impulses. For example, Hardesty built up a model that he thinks resembles the membrana tectoria, and he demonstrates that it actually responds in different parts to tones of different pitch. Recently an Englishman developed a model that, he assumes, demonstrates what the responses of the membrana basilaris would be, provided it responded according to the principle of physical resonance. Ewald developed a model to show how undulations throughout the membrana basilaris represented the type of responses of this membrane to sound impulses.

My feeling is that any one who has studied the actual structure of the organ of Corti through the microscope would hardly feel like attempting to create any model that could be taken seriously as demonstrating how structures in the cochlea perform their function. Hardesty's model, for example, no more resembles the membrana tectoria than does a tapering haystack; Ewald's model bears no more resemblance to the membrana basilaris than does a stretched sheet, and the model that I referred to, demonstrating the response of the membrana basilaris according to the principle of physical resonance, demonstrates only what we all knew before, that such a model should respond to the principle of physical resonance, and is not a demonstration of what the membrana basilaris might do.

Dr. Pohlman points out objections to Ewald's theory but fails to mention the most fundamental of all objections, namely, that the attachment of the hair cells to the under-surface of the membrana tectoria makes it impossible for certain groups of hair cells occupying the crests of supposed wavelike undulations in the membrana basilaris to be stimulated separately. The anatomic relations between the hair cells and the tectorial membrane make it necessary for every undulation of the membrana basilaris to stimulate not only the hair cells that occupy the crests of these undulations, but all the hair cells throughout Corti's organ and would thus, as Ewald himself points out, render peripheral tone analysis impossible.

I think these discussions of the principles underlying the physiology of hearing are interesting and more or less profitable, but not in the sense that they solve for us in any satisfactory manner any particular clinical problems. I agree with Dr. Pohlman that operations aiming to open a passage for sound impulses to enter the labyrinth by boring through the labyrinthine wall can hardly be expected to give any satisfaction. In the first place, to carry out an operation for making a fistula of the bony capsule of the labyrinth without breaking through into the cavity and allowing the escape of perilymph and endolymph is, to say the least, a precarious undertaking, for if the fluids of the labyrinth escape, the function is entirely destroyed. In the second place, it must be an exceptional situation in which an opening made in the bone will not close spontaneously and thus destroy any possible good effects from the operation. And finally, the operation would be undertaken only in cases of profound deafness, and in these cases the deafness is not so much the result of fixation of the stapes itself as of secondary degeneration of the nerve, which cannot be benefited by making a fistula into the labyrinth. My feeling is that if the facts were placed squarely before a patient on whom we proposed to undertake an operation of this kind, he would decide not to have it done.

DR. ALFRED LEWY: I once listened to Einstein expounding his theory, and I thought I caught a glimmer of it. Later I heard the theory explained by Professor Lunn at the University of Chicago, and I thought I had gotten a little more out of it. But when I tried to tell anybody else what I thought I understood, it was impossible to do so.

I feel very much the same with respect to Dr. Pohlman. I heard most of what he said this afternoon and this evening, and I was so entranced in listening to what he had to tell us that I forgot to put down any notes until after he got through speaking. However, he apparently demolished most of the theories of the perception of sound. For some reason, he did not touch on the old and perhaps now discarded telephone theory. I should like to have him, if he can, tell us what is the matter with that. We have been told a number of times what is the matter with it, but I should like to have his version.

Rather than discuss his paper, I shall ask him a few questions. As I indicated, I do not feel competent to discuss it. How does he know what the range of hearing in the parrot is? I know that he judges it by what he calls the motor output, by which I think he means the range of sound that a parrot is capable of making. But it has been found possible to obtain in animals certain reflexes of the eye or some other action that would indicate whether they do or do not hear a certain sound. I should like to ask Dr. Pohlman whether that has been

carried out in connection with the parrot or with any other animal in which the structure of the cochlea is different from that of the human being.

Not being a physicist, I should like to ask whether the mechanical vibrations that have been transmitted through the mechanism of the ear may have an electrochemical effect on the nerve endings. The exposition of a phylogenetic history of the cochlea raises the question whether some of the original neuro-epithelium may be attuned to an electrochemical effect that is connotated by that particular pitch. The later developments may be attuned to a different pitch.

Dr. Pohlman promised us this afternoon that he would tell us what his theory is. I did not understand that he expounded any theory of his own. I have always been troubled in understanding exactly why sound waves are called waves. If they are areas of expansion and rarefaction, contraction in the air, why, in the teaching of the theory of sound waves, does one not draw a picture of an interrupted series of dashes of equal length to represent frequencies and of different dashes of different degrees of thickness to represent different degrees of energy, in other words, greater amplitude? I do not know, but it seems to me if it were explained in that manner, it would be a little more easily grasped.

DR. ROBERT SONNENSCHEIN: We have listened to a great many fundamental and important facts regarding physics and, to a certain extent, the physiology of hearing. The various theories of hearing I know very little about. I know the names of some of them: Helmholtz' theory of peripheral analysis, the telephone theory, the theory of central analysis and volley, and others.

It seems to me that the highly scientific aspects of the subject that have been stressed perhaps warrant calling attention to a few of the points that Dr. Pohlman mentioned. He mentioned that there are certain unreal phenomena such as paracousis willisii. My attention was called by the essayist to the first edition of Troeltsch's book, which appeared in 1862, in which the author stated that the person who is hard of hearing does not feel the masking effects of the loud sounds about him at the same pitch at which he is deafened; when the speaker, raising his voice because he feels the masking effect of these sounds, speaks much louder, the person with the impairment of conduction hears. Dr. Shambaugh did some excellent work on paracousis with the audiometer. I think it is important to stress that point, because one still finds excellent otologists with divergent opinions. No less an authority than Professor Alexander said that he could prove, on the street car and in other places, that paracousis is an actual phenomenon.

The point has been brought out that the person who apparently has increased bone conduction, at least what is called prolonged bone conduction, when tested in an office does not have increased bone conduction when tested in the silent chamber. Dr. Pohlman has shown that there is such a thing as actual increase in bone conduction when one loads the drum membrane or occludes the external auditory canal.

I have always maintained that it is not the best policy to test the patient with one's own bone conduction. That presupposes that one's own bone conduction is normal. It is better to have an objective standard for use in these tests, determined by examination of, say, 100 apparently normal persons. If the bone conduction varies fifty seconds, for instance, when used in a certain manner, then one can know, from practical experience, that when a person hears only thirty seconds his bone conduction is diminished. If the next subject hears one hundred seconds, the conduction is increased.

It does not make much difference whether bone conduction in a particular case is increased from the scientific standpoint, because otologists, as a rule, must examine patients in noisy places. Therefore, if one does the tests in a certain manner and knows the usual results, one has the criteria for estimating the degree of conduction from a practical standpoint.

The absolute test of bone conduction, about which we read a good deal in the English literature, is performed by placing the finger in the ear and producing the prolonged bone conduction by occluding the auditory canal. This is based on

the same principle as one type of the old Bing test. In normal persons bone conduction is increased, but in a person with a negative Rinne reaction and lengthened bone conduction, occlusion of the ear no longer increases the bone conduction. I mention this point because there is so much confusion on the subject.

Dr. Pohlman, if I quote him correctly, stated in an article published last year that the apparatus for the transmission of air sounds appears to be of equal efficiency throughout the audible scale. If a person hears sounds of any frequency at normal intensity, he may be regarded as not having conduction deafness. Of course, that is true, as is also his statement that if when one tests a person with the audiometer he has diminished hearing for the low tones, he also has diminished hearing for the high tones. If he has impairment of conduction, he also does not hear the high tones well. That, no doubt, is true when finer quantitative measurements are involved. But if in actual practice it does not prove true, one is not testing accurately, but testing in a practical manner, by means of the voice. And, as has been stated so often, even though the voice cannot be standardized as regards intensity or pitch, especially intensity, and different voices vary qualitatively because the overtones in the speech give the peculiar quality to different persons, nevertheless, if the test is used in the proper manner, valuable results are secured. For instance, if one has a patient stand 20 feet (5 meters) away and whispers in low tones, the patient may not hear the low tones until the examiner is within a meter of him, while if high pitched tones are used he often hears them at a distance of 5 or 6 meters. We know that such a condition is a beginning impairment of conduction. The high tones when tested with the audiometer may show sc ne contraction. But those are the very tones about which Dr. Pohlman spoke, which we are not conscious of hearing.

I am as much interested as any one in knowing scientific facts, but one should bear in mind that the various theories of hearing are not only not settled, but in greater confusion than ever before. Every year we are getting modifications of the theories of hearing, just as is the case in therapeutics, in which ten, fifteen or twenty remedies are suggested for a certain disease. But one may be sure that no remedy is very efficient, because if it were, physicians would use that one exclusively. In the treatment for syphilis, one relies on mercury, arsenic and the iodides, instead of using twenty or thirty different medicaments.

DR. J. HOLINGER: It seems to me that Dr. Pohlman is going too far in rejecting every attempt at explanation of the act of hearing. This nihilism is unfortunate, because it creates in many persons a state of mind that says, "What is the use of bothering?" and it paralyzes interest and real study. This is especially unjustifiable at the present time when the study of many observations on the battle-fields of the Great War shows so many coincidences with the results of experiments made on animals fifteen and twenty-five years earlier. I cannot understand how anybody can doubt the interaction between the tectorial membrane and the hair cells when it is shown in experiments with overirritation of birds' ears that the tectorial membrane can crush and smash hair cells, and that only those that it can reach are affected, while those beyond its reach are intact. All these points have been discussed before this society so many times that I feel that most of the Chicago otologists know the point beyond which they cannot follow Dr. Pohlman. For example, the tectorial membrane is a tissue floating in the mobile fluid of the labyrinth and a little more viscous than the fluid itself, and is therefore very apt to react to every movement of and in the fluid. If one tries to understand the experiments on birds from this point of view, the effect of the round window appears in another light. The interior of the labyrinth is elastic, not inelastic as Dr. Pohlman says. This changes the attitude of the otologist as to the function and action of the round window. Deafness, as well as intact hearing, has been found in ears in which the round window was more or less immovable. Clinical experiences are certainly not final, and to state conclusions beyond what has clinically been established is putting too much weight on uncertainties.

DR. A. G. POHLMAN: The function of the auricle in the human being appears to be restricted largely to reflection of the higher frequencies, and its efficiency seems to be greatest at or above 8,000 Hertz waves. Increasing the size of the auricle by placing the hand back of the ear lowers the frequencies that it will reflect efficiently. One sees the effect of the auricle in tests at high frequencies with the audiometer, in which the patients say that they hear a given tone better when the receiver is removed a short distance from the ear.

I neglected to mention a point that may be of interest to the otologists. When a patient is tested with the audiometer and a curve is established, one may conclude that this person has lost a certain percentage of his useful hearing. Useful hearing, however, to the patient usually means useful hearing so far as conversational purposes are concerned. While a great deal of work has been done with reference to the masking effect of one pure tone on another pure tone, the experimental aspects of a masking effect of low frequency components on the high frequencies in a complicated sound, as in speech, and the effect on the intelligibility have not been accurately determined. The audiometric curves are therefore not necessarily a criterion of the functional efficiency so far as the spoken voice is concerned, without more definite evaluation.

Experimental production of degeneration of the cochlear end-organ has been repeatedly accomplished. It is, however, reasonable to suppose that the auditory cells undergo fatigue just as do any other highly organized cells. To say that a given area of the cochlear end-organ has degenerated on account of a prolonged exhibition of a loud sound does not mean that either the basilar membrane or the tectorial membrane has been mechanically responsible for the lesion. The cell area might have degenerated through excessive stimulation, irrespective of how this stimulation was brought about. The evidence is, therefore, not conclusive that the activation of the auditory cells is extrinsic.

Dr. Shambaugh referred to the development of the otolithic membrane and otoliths. It must not be forgotten that the functional relation of the otoliths to the otolithic membrane is one of displacement to stimuli of slow motion, and I fail to see how the tectorial membrane without otoliths might react to high frequency vibrations of extremely small amplitude.

Dr. Lewy asked about the telephone theory, which in my opinion is not a theory at all, because it explains nothing and assumes a central analysis that is seemingly contrary to the facts. If one carbon button or a single condenser transmitter can handle practically all audiofrequencies through a wide range of intensity, it is difficult to see why nature should have supplied the internal ear with, say, 20,000 auditory cells, unless a peripheral analysis is indicated. Rutherford, like other physicists, may have proposed his conception with no very definite ideas on either the morphology or the physiology of the apparatus.

As to the range of hearing in birds, it may be that the Weaver and Bray technic will establish this quite definitely. I believe one must admit that the mechanics of hearing in birds must be similar to that in mammals. A mocking bird that imitates the creaking of a rusty hinge must hear at least about 10,000 vibrations if it is reproducing what it hears; otherwise the creaking noise would not sound quite normal to a human being. It has been demonstrated that the noise produced by the jingling of a bunch of keys changes in quality if vibrations above 8,000 are filtered out. If the mocking bird can imitate the creaking of the hinge of a gate, I for one am prepared to assume that it hears at least the frequency range of the human being.

To say that the sound produces an electrochemical effect in the auditory cells is in my opinion merely giving a name to an unknown quantity. If I knew what is meant by an electrochemical effect I might be able to agree or to disagree with the proposed solution.

I believe that I demonstrated that all that was necessary to cut down the resonant response in a resonator was to change the weight of the contained gas and indicated that there was nothing to see. It is accordingly not necessary to believe that one must visualize the adaptations to audition, and perhaps one must hear them to understand them.

So far as my own theory of hearing is concerned, I cannot say that I really have one. My guess is that there is a peripheral analysis on the part of the auditory cells, and that discrete frequencies are resolved serially, arranged from base to tip with highest frequencies at the base. I have no quarrel with either the Adrian explanation of the Weaver and Bray experiments on the basis of a microphonic action of the cochlea or the explanation suggested by Weaver and Bray that the action currents in the auditory nerve reproduce the frequencies found in a composite source. So far as the mechanics of the system is concerned, both may be expressions of a similar effect. The latest experiments on turtles indicate that they do not hear sounds above 1,000 Hertz waves, which conforms with the proposal that a progressive development takes place toward the high frequency resolution as the form evolves.

Sound vibrations are spoken of as waves because they may be visualized as transverse displacements rather than as longitudinal displacements. The "to and fro" swing in the molecules in reference to the wavelength is readily shown in a transverse wave form. The sound advances somewhat as a soap bubble distends.

Dr. Sonnenschein indicates that it would be a good idea to standardize the fork for bone conduction and make it unnecessary to include the observer in the test. This is quite correct, provided the standardization on normal persons is done under conditions of silence, because otherwise the readings will all be too low owing to the masking of adventitious noises. Tests on persons with a negative Rinne reaction who have normal perception apparatus do not show prolonged bone conduction. I am not concerned with the practical application of the test so long as we agree that prolonged bone conduction does not occur, except in persons with normal perception apparatus and with a functional sound conduction system under conditions in which the drum membrane is loaded or the external canal occluded.

DR. ROBERT SONNENSCHEIN: In the clinical application, even if the premises are wrong, one has the criterion of comparison.

DR. A. G. POHLMAN: We are dealing with the mechanics, not with somebody's conception of the results obtained under unsatisfactory conditions of testing. I have agreed that a conduction-deafened person with even depression in acuity throughout the audible range will show relatively greater disability at the low frequency end of the audible range of sound. This, as I explained, is a function of the internal ear and is not dependent on the mechanics of conduction. I do not agree that in frank conduction deafness the low end shows greater losses in sensitivity than the high end of the audible range.

The theories of hearing, as I have tried to indicate, are nothing more than darns on the same old sock, and what we need is a new sock. Dr. Holinger objects to my attempt at wiping out all theories and starting in all over again. The tectorial membrane, as Dr. Shambaugh has demonstrated, is applied to the ciliae of the auditory cells just as the otolithic membrane is applied to the cells of the vestibular apparatus. If the cochlear apparatus is developed on the fundamental pattern of the elements of the otic vesicle, there is no reason why it might not take on the pattern of the vestibular system without definite function for either the tectorial membrane or the ciliae. Ciliae have persisted elsewhere without function. Structures do not disappear in the body merely because they are not used, but because their persistence may in some way interfere with selection. There is on the abdominal wall a muscle, the pyramidalis, which has only one function, namely, to open the marsupium. People are just as fit to survive without a marsupium, and the persistence of the muscle may be accounted for on the basis that it does not interfere with survival.

Dr. Holinger has mentioned instances of occlusion of the round window in the human being. He probably referred to the two cases reported by Oppikofer in which the niche was entirely filled with fatty tissue. While the tests of acuity made by Oppikofer are not entirely satisfactory from an audiometric standpoint, it is worth noting that he reported the two cases as being associated with normal acuity for air-conducted sounds.

The experiments of Hughson and Crowe have suggested to them the function of the round window as a shock absorber, although no explanation of how this effect might be produced is given. It appears that the round window may act as a shock absorber only by having its vibrations in phase with those of the oval window. Under this condition the action would be to suppress transverse vibrations at the basilar membrane. However, the evidence is that a cotton plug against the round window affects the labyrinth through its vibration, not its pressure, and if transverse vibrations are demanded in the cochlear duct for audition, then the person should hear less well with the prosthesis in place. The evidence shows increased acuity.

In answer to the question on the model reactions, every one knows that the size, viscosity of the liquid, caliber of the tube, etc., influence the result. The pattern developed in the apparatus mentioned was apparently not influenced by the length of the column of liquid, and a pattern was developed only when the oscillations were from two to three per second. Even at five to the second, the pattern disappeared. We must also not forget that the air amplitude in the external auditory canal at minimum audition is a small fraction of a millionth of a millimeter, and the chances are that there is considerable loss in amplitude as the displacement is transmitted to the liquid of the labyrinth. It may be that this displacement must be expressed in terms of hundred-millionths of a millimeter. We are dealing with measurements that split the size of a molecule. The displacements probably pass through everything — basilar membrane, tectorial membrane, auditory cells and even the bony wall of the container. If Dr. Holinger believes displacements of this order can have an effect in the interaction of the ciliae and the tectorial membrane, it is his privilege, just as it is my privilege not to believe this possible.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLARYNGOLOGY

H. P. Schenck, M.D., *Reporter*

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Ralph Butler, M.D., *Chairman*

Cerebrospinal Rhinorrhea: Report of Two Cases. Dr. J. C. Donnelly (by invitation).

Two cases of cerebrospinal rhinorrhea were reported: in one, which was spontaneous, the patient survived; in the other, which was traumatic, the patient died. The first case, that of an asthmatic man, aged 57, presented advanced hyperplasia of both middle turbinates, and the middle meatuses and the left spheno-ethmoidal area were filled with polypi. The frontal and right maxillary sinuses were hazy on transillumination; the right antrum was opaque. As the pathologic condition of the sinus was thought to be an important factor in the bronchial asthma, bilateral ethmoidectomy was undertaken. When the right middle turbinate was removed, extensive polypoid degeneration was found in the posterior ethmoidal cells. Some superficial tissue was removed with a Blakesley alligator forceps. The second bite into the polypoid mass was followed by a thin, watery discharge, which became more profuse and was noted to pulsate. As the leakage was thought to be cerebrospinal fluid, operation was stopped and the patient sent to bed. While being placed on the ward carriage, the patient fainted. On the basis that this was due to a sudden decompression and that the loss of fluid was too great, the ethmoidal area was packed with iodoform gauze. Epinephrine hydrochloride and atropine were administered, and in a few minutes the patient regained consciousness.

On his return to the ward, the patient was cold and clammy, with a temperature of 97.2 F. and a pulse rate of 60. The nasal packing was then removed, and a policy of dehydration was instituted to reduce intracranial pressure to facilitate early closure of the operative wound. To this end fluids were restricted, and large doses of magnesium sulphate were given. Twenty-four hours after operation, the rhinorrhea ceased. Two days after operation, the temperature and pulse rate remained normal, and the patient's strength was such that he repeatedly requested to go home. Seventy-two hours after operation, the temperature soared to 103 F., and the patient complained of severe occipital headaches, chills and nausea. Restlessness and delirium followed. The pupils contracted, but reacted to light. A spinal puncture revealed turbid fluid but no increase in pressure; the cell count was 16,000 polymorphonuclear cells. Kernig's sign and cervical rigidity appeared, and the cell count rose to 35,000. Five days after operation, the patient died.

At autopsy, the surface of the brain was found to be covered by a thick, yellowish, purulent exudate, which was especially abundant toward the base. The convolutions were distinctly flattened. Hemolytic streptococci were cultured from the cerebrospinal fluid both before and after death. An opening with ragged edges, measuring 1 by 1.5 cm., was found on the right ethmoidal roof, about 1 cm. from the midline or 1 cm. from the lamina cribrosa. This opening led into the ethmoidal cells, which were filled with a soft, foul-smelling, gelatinous mucosa. There appeared to be senile atrophy of the bone, but no necrosis. The left ethmoidal roof was likewise atrophic.

The second patient, a woman of 60, weighing 225 pounds (102 Kg.), had noticed a watery discharge from the right side of the nose during an attack of influenza. Recovery from influenza was uneventful, but thereafter she awoke every morning with a severe bitemporal headache. At 9 or 10 o'clock, the headache disappeared and rhinorrhea began. The flow continued intermittently throughout the day and night, with the exception of the early morning hours, at which time her headache was present. Approximately 12 ounces (355 cc.) of fluid was lost in twenty-four hours. Bilateral pressure on the internal jugular veins occasionally produced a gush of fluid from the nostril. Other nasal symptoms were absent, and there was no history of injury or operation.

A clear, watery fluid was observed to ooze from the right sphenoidal ostium. A roentgenogram revealed a peculiar appearance of the middle fossae, the identity of which was uncertain. The dorsum sellae was quite thin, as if eroded from behind. This may also have caused an erosion into the sphenoidal sinus and would thus account for the cerebrospinal rhinorrhea. The only therapy of proved value has been a reduction in the amount of fluid intake. At the time of presentation, it was twenty-one months since the onset of the rhinorrhea, which continued, but the patient was attending to her household duties.

DISCUSSION

DR. GEORGE M. COATES: These cases are rare and infrequently reported. It is disturbing to the surgeon operating on the ethmoid to find that the roof of the ethmoidal structure may be broken by a small tug. We usually suspect a break in the cribriform plate, but in the traumatic case there was an unusual break. When meningitis occurs after ethmoidal operations, I often suspect that the infection may travel by way of the perineural sheaths. In one of my cases in which there was pituitary enlargement, a typical case of acromegaly, the patient had cerebrospinal rhinorrhea for seven years, and is now alive and well. Several drops of fluid are lost per minute, and the pathologist found this fluid to be typical of cerebrospinal fluid.

DR. JOHN E. LOFTUS: Such accidents must occur to all of us. They are rarely reported. I think it is a mistake to pack after ethmoidal operations should rhinorrhea appear. A patient operated on in Germany had a meningocele removed in mistake for a nasal polyp. When the mistake was recognized, immediate opera-

tion was done to suture the dura, and the patient got well. Cases of spontaneous cerebrospinal rhinorrhea have the same type of history. Patients with tumors and internal hydrocephalus do not always develop cerebrospinal rhinorrhea. The cases of tumor in which Dr. Cushing performs transnasal sphenoidal operations all heal without the development of rhinorrhea.

DR. WARREN B. DAVIS: Any of us who have worked for a long time in the dissecting room recall sphenoids in which definite dehiscences of bone have occurred. Usually they are posterior and lateral or in the superior portion of the sinus. These areas become weak because of pressure. In a series of sinuses we studied several years ago, we found thinning of the bone in a number of sphenoids.

DR. GEORGE B. WOOD: A colored woman with traumatic cerebrospinal rhinorrhea was admitted to the Graduate Hospital several years ago. She had been struck several times by a hammer in the hands of her husband. The first blow destroyed the lower wall of the frontal sinus and crushed the nasal bone and the anterior ethmoidal cells; the second blow broke and depressed the anterior wall of the antrum; the third blow cut off the external ear and depressed the mastoid area. A profuse cerebrospinal leak developed from the cribriform plate. Dr. Grant cut across the brow, turned back osteoplastic flaps, elevated the frontal lobe and explored the cribriform plate. The site of the leak could not be distinguished, so packing was placed over the cribriform plate. The woman got well, and the cerebrospinal rhinorrhea disappeared. If we are daring enough, we may save some of these patients.

DR. SAMUEL SKILLERN: The posterior portion of the ethmoid is the dangerous area. Several years ago I opened the dura. I immediately made an incision through the anterior and posterior plates of the frontal sinus, found the hole, packed and waited. The patient died within twenty-four hours. In another case, in which I attempted to sound the sphenoid sinus after a complete extirpation of the ethmoid on the right side, there did not seem to be a posterior sphenoidal wall. I obtained a longer probe, but this did not touch a bony wall. After calling in my brother and Dr. Ridpath, we placed a longer probe through the ostium, and an x-ray plate showed the probe touching the top of the cranium. I expected the usual fatality in forty-eight hours, but the patient made an excellent recovery and was discharged from the hospital. A year later a splitting headache and delirium developed, and the patient died. At autopsy, when the skull cap was removed, from 4 to 8 ounces (118 to 236 cc.) of blood gushed forth. We found a hemangioma of the left frontoparietal lobe, which had burst. The Wassermann reaction of the blood was reported as 4 plus. A probe could not be pushed through the dehiscence, and no vestige could be found of the opening through which the probe had previously been introduced.

SERIOUS COMPLICATIONS FOLLOWING TONSILLECTOMY. DR. GEORGE B. WOOD.

A boy, aged 4 years, was admitted to the hospital, the chief complaint being swelling of the left knee beginning ten days after vaccination against smallpox. A slight irregular fever continued until the knee was incised, but five days after operation the temperature became normal. Three months after admission, tonsillectomy was considered with a view to removing all sources of infection before discharging the patient. A rise in temperature to 102 F. for twenty-four hours at this time was due to a slight infection of the tonsils. The tonsils were large, but were otherwise normal. The paranasal sinuses were normal. Fluoroscopic examination of the lungs showed a slight increase in density in each hilus, but not sufficient to suggest tuberculosis, and the von Pirquet test gave negative results. Fluid from the knee was sterile and failed to kill an inoculated guinea-pig. The white blood cell count varied from 7,000 to 15,000, with a slight excess of lymphocytes. The urine was normal.

In accordance with the usual technic, sodium bicarbonate was administered for twenty-four hours before operation, with a diet high in carbohydrate, the teeth

being cleaned thoroughly and moderate evacuation of the bowel being induced. The tonsils and adenoid tissue were removed under ether anesthesia, and the child left the table in good condition, with a dry throat and a pulse rate of 112. Three hours later the temperature had risen to 102 F. Fifteen hours after operation it had reached 104 F., and three hours later it was 105 F., later reaching 106.2 F. The pulse rate increased as the temperature rose, its highest rate being 150. There was no active bleeding after the operation. The child was rational until two hours before death, which occurred twenty-four hours after operation. Just before death, the child became cyanotic. There were no definite signs of pneumonia. The throat showed a certain amount of edema about the uvula, and the patient complained of a good deal of pain in it. There were no neurologic symptoms until an hour and a half or two hours before death, when rigidity of the neck and spine appeared. No autopsy could be obtained.

The cause of death in these cases of severe postoperative hyperpyrexia is extremely difficult to explain. There is a close resemblance between these cases and those in which there is a lesion of the diencephalon, in which theoretically the heat center is located. There are clinical examples in which attempts at removal of suprasellar tumors that encroached on the base of the third ventricle were followed by a gradual increase in temperature, with death within two or three days. Also, in certain cases of fracture at the base of the skull, there is marked hyperpyrexia. Further, hemorrhage into the third ventricle or an embolism that involves this area of the brain would produce a similar clinical picture. It is scarcely conceivable that any surgical trauma in the region of the faucial and pharyngeal tonsils could in any possible way directly injure the brain stem. As far as the blood vessels are concerned, there is no direct connection between the pharyngeal vault and the suprasellar region, so that the embolic theory is scarcely tenable. The only possible anatomic connection would be perhaps a persistent Rathke pouch, the embryologic anlage from which the anterior portion of the pituitary body is formed, originating from the nasopharynx. However, these cases of postoperative hyperpyrexia are not always results of operations on the tonsils; they have been noted in children following a variety of operations.

SUDDEN DEATH ELEVEN HOURS AFTER TONSILLECTOMY FOLLOWING THE LOCAL APPLICATION OF COCAINE IN AN EFFORT TO CHECK BLEEDING. DR. KARL M. HOUSER.

This article will be published in full in a later issue of the ARCHIVES.

SERIOUS COMPLICATIONS FOLLOWING TONSILLECTOMY. DR. EDWARD H. CAMPBELL.

In a woman, aged 35, cavernous sinus thrombophlebitis developed following tonsillectomy under local anesthesia. The anesthesia consisted of an infiltration of the peritonsillar area with 1 per cent procaine hydrochloride solution that contained a few drops of epinephrine. The tonsils were removed by the dissection and snare method. The day following operation the temperature was 100 F., but there were no other unusual symptoms at this time. On the second day after operation, a severe chill was followed by a rise in temperature and a swelling in the right peritonsillar region suggesting an early abscess. The abscess was incised the following day, with the evacuation of thick, greenish pus containing gram-positive staphylococci and gram-positive diplococci. The temperature at this time varied from 99.4 to 100.3 F., and the leukocyte count was 17,700, with 82 per cent neutrophils.

The symptoms in the throat improved after the incision, but the following day pain and tenderness developed in the left temporal and parotid region. Twenty-four hours later edema, first of the right eye and then of the left, was noted. Tenderness and swelling of the left side of the face increased and extended downward over the parotid region, but no fluctuation developed. There was swelling

in the left peritonsillar region, but not sufficient to indicate an abscess. There were daily chills followed by a moderate rise in temperature, and a blood culture at this time contained nonhemolytic streptococci. Swelling of the eyelids rapidly increased, and a distinct proptosis was evident. Paralysis of the external rectus muscle on the right was noted, and other ocular movements were faulty, but no swelling of the optic disks or hemorrhages occurred. Ten days after operation, the swelling of the left side of the face had developed to such an extent that, although no distinct fluctuation was evident, a deep incision was made in the preauricular area, releasing a small amount of pus and very dark colored blood. The following day the incision was enlarged and deepened, but an abscess pocket was not found. General stimulation, frequent infusions of dextrose and saline solution and blood transfusions were employed, but the ocular signs of cavernous sinus thrombosis steadily progressed. Both eyelids became more edematous, the exophthalmos steadily increased, considerable ecchymosis developed, and the eyeball became immovable. The temperature reached 107 F., and the patient died on the thirteenth day after operation.

A partial autopsy was permitted, and the incision on the left side of the face was explored. A considerable amount of seropus was expressed from the tissues, but no localized abscess pocket could be found. Sections of the lungs showed multiple hematogenous abscesses; the heart and liver showed cloudy swelling, and the kidneys gave evidence of acute tubercular necrosis. Unfortunately, the brain could not be obtained, but the typical signs and symptoms of cavernous sinus thrombosis (thrombophlebitis) plainly indicated the diagnosis.

DISCUSSION ON ARTICLES BY DRS. WOOD, HOUSER AND CAMPBELL

DR. B. H. SHUSTER: One of my patients, a baby 2½ years of age, barely escaped death after tonsillectomy. The child was taken home the day after operation, and later in the day I was called and told that the baby was falling asleep and could not be aroused. No food had been taken for a day and a half. A catheterized specimen of urine showed sugar and acetone. The patient was filled with fluids, and dextrose and alkalis were administered; the patient survived. Dr. Campbell's case probably came about by thrombosis by way of the pterygoid plexus, but there may also be thrombosis of the internal jugular vein.

OSTEOMA OF THE PARANASAL SINUSES: CASE REPORT. DR. J. AUSTIN SMITH.

A boy, aged 16 years, came to the Jefferson Clinic complaining of marked swelling, redness and tenderness over the left frontal sinus. At this time the left eye was closed by the swelling and edema of the surrounding structures. A small, hard lump on the left side of the nose at the inner third of the floor of the left frontal sinus had first been noted by the patient two years before coming to the clinic. It was not tender at that time and gave him no discomfort, but it gradually increased in size, and later there was swelling of the forehead and the lump encroached on his eye. A month before coming under observation, the patient was in a fight and was struck heavily at the site of the growth.

On examination, the patient presented the picture of acute frontal sinusitis with perforation and involvement of the soft parts. The eyelids were closed by the surrounding swelling and edema, and there was marked tenderness with fluctuation over the swelling above the eye. The nasal chambers were clear, and ventilation and drainage appeared good. To secure drainage, an incision was made beneath the left brow, and considerable pus was obtained. The cellulitis subsided until the tumor could be readily palpated. No attempt was made to remove the growth. Roentgenographic studies revealed a lesion involving the left frontal sinus in which there was a mass with a density almost that of bone. It apparently projected into the orbit. The appearance was that of an osteoma. The direct postero-anterior films showed that the left frontal sinus was completely filled with the tumor, and that it extended into the right frontal sinus as well as into the left orbit. (Roentgenograms and the patient were presented.)

DISCUSSION

DR. ROBERT J. HUNTER: I have a patient under observation who came to our attention four years ago. The tumor appears more dense in the roentgenograms than does the one reported, and it measures from about 2 to 3 mm. in diameter. The patient, a young lady, returned because of a mild cold. The osteoma appears to be the same size as it was four years ago.

DR. WARREN B. DAVIS: About six or eight years ago, Dr. Shallow asked me to assist him in an operation on an osteoma of the orbit, involving the ethmoidal cells and the antrum. There was no sinus infection. Through a radical frontal incision, a mass of dense bone was discovered, which was rather spherical and from about ¾ to 1 inch (1.87 to 2.5 cm.) in diameter. The eyeball was displaced to facilitate removal of the tumor, and the lower portion of the orbital plate and the ethmoidal plate were removed. The eyeball returned to its position, and there has been no recurrence to date.

DR. PHILIP STOUT: I saw a case of osteoma of the frontal sinus about a year ago. Six months later a roentgenogram showed it to be about the same size as formerly. These tumors sometimes respond to irradiation.

H. P. SCHENCK, M.D., *Reporter*

Nov. 17, 1931

RALPH BUTLER, M.D., *Chairman*

A SHORT RÉSUMÉ OF THE RESULTS OF THE BALLANCE-DUEL RESEARCH IN NERVE ANASTOMOSIS AND THE EMPLOYMENT OF NERVE GRAFTS IN THE SURGICAL TREATMENT OF FACIAL PALSY (moving pictures and lantern slides). DR. ARTHUR B. DUEL (by invitation).

Sir Charles Ballance performed his first anastomosis of the hypoglossal nerve to the facial nerve in 1895. His important work on the anatomy and surgery of the temporal bone was widely recognized twenty-five years before this meeting. A year after the World War, he came to the United States and during his visit suggested to me that we retire to a quiet place where animal experiments could be carried out to devise an operation for the cure of facial palsy without associated movements to mar the result. A laboratory was built in the country which eventually accommodated forty large apes, from seventy to eighty cats and twenty-five large Louisiana frogs. Sir Charles Ballance superintended the building of the laboratory and when that was completed confined himself entirely to the work in hand, working with marvelous industry from day to day, without exercise or sufficient rest.

Various types of anastomoses were considered. Patients with an anastomosis of the spinal accessory and facial nerves were unable to use the facial muscles without accessory movements, but a large number of similar anastomoses were done to verify previous experiences. Anastomoses of the descendens noni, glossopharyngeal and chorda tympani nerves with the facial nerve were attempted. In every instance in which such anastomoses were done, the facial canal was opened, plugged with gold and carefully inspected, because in Sir Charles Ballance's experience, the stump of the facial nerve was in such close proximity to the anastomosis that there was a question whether or not tissue had leaped across the scar by residual strands and could thus be suspected of influencing results. In the smaller animals the descendens noni and the hypoglossal nerves were anastomosed with the facial. Then the auriculotemporal nerve was joined to the facial in larger animals, a nerve being utilized that could show no associated movements,

but this failed to produce the finer movements obtained when the hypoglossal or spinal accessory nerves were used.

For a long time, Sir Charles Ballance had advocated taking the facial nerve out of its canal and running it across the tympanum to get length. The technic required to accomplish this without trauma was carefully developed. The use of the great occipital nerve for anastomosis with the facial precluded associated movements. For this reason the great auricular nerve was likewise tried.

In one infant, in whom the facial nerve had been largely destroyed by a mastoid operation, one end of the nerve was situated in the parotid gland and the other end presented at the geniculate ganglion. Recovery followed the grafting of the facial proximal portion to the facial distal portion. In a woman 16 mm. of the facial nerve had been removed in a radical operation, resulting in complete palsy. A portion of the nerve of Bell was placed in the dehiscence four and one half months after operation, and this patient now has some ability to close the eye.

A baboon in which the intercostal nerve was used for anastomosis with the facial nerve showed complete recovery. In another patient, the nerve of Bell could not be located at operation, while a large cutaneous nerve was uncovered and an attempt was made to connect it with the facial nerve. Its length was not sufficient, but it was thought that it would work out as well as a motor nerve. Making certain that the distal portion of the nerve was alive, a portion of the third intercostal was placed in the canal, but its length appeared insufficient. A piece of the fourth intercostal nerve was then taken and placed alongside the fragment of the third intercostal nerve and the whole was covered with dental gold. Six days later, the patient obtained the sensation of movement.

No one can predict the degree of recovery in these cases. The animals used in this series of experiments with grafts have shown recoveries that are practically perfect. Twenty-four animals have shown recovery from dehiscences of from 1 to 16 mm. Motor and sensory nerves were both used, with an equal amount of recovery. A child aged 8 months shows recovery which is progressing; at this time the normal expression of pleasure or crying is almost perfect. If a man were like an animal, almost perfect recovery could be obtained; in man it is difficult ever to restore a divided or injured facial nerve and obtain a result that synchronously shows the higher emotions. It is difficult to get the finer gradations of expression through a repaired nerve. In order to find the anastomotic tissue, one gets trauma and scar tissue and therefore produces interference with synchronous effects. Fear and laughter may be well expressed, but the finer gradations of emotion will be different from those obtained before injury occurred.

The operation as perfected will restore the facial muscles and avoid the associated movements, which in other operations can be eliminated only at best by training. In addition, this operation avoids a disfiguring scar in the neck and the loss of function of other muscles. Any anastomotic operation is difficult and this is no exception. No one will be able to do it without taking time enough to master the technic.

DISCUSSION

DR. CHARLES H. FRAZIER: It is at once stimulating and refreshing to hear of this distinct achievement. The operation described here has been performed on man and animal, and a treatment has been elaborated that is a distinct step forward in the therapy for facial paralysis. The repair of damaged nerves is not unfamiliar to me. In one center during the war we had 1,200 such cases and I felt rather fed up on operations involving the peripheral and cranial nerves. I should like to confine my remarks wholly to facial paralysis, and to review some of my observations. When should the operation be performed after facial paralysis? Our rule is to wait six months, but one cannot be too arbitrary. One day after six months one of my patients had a quiver at the angle of the mouth. Another patient, a woman with a tumor of the parotid, began to recover one year after operation. A peculiar sensation foretells recovery; a sense of power appears before the patient is able to demonstrate power. The time of operation depends on the lesion, i. e., whether the injury is due to a mastoid operation, tumor or

other cause. Another problem frequently presented is: "How long can an operation be deferred?" In one case twenty-two years had elapsed before operation, which was followed by reasonable success. I always tell my patients what they may expect if we use the hypoglossal or intercostal nerves: that they will have facial expression and will be able to close the eye, but will not be able to use the occipitofrontalis; this function is usually the last to appear and sometimes never returns. I also tell patients that if they take the time and the trouble, they can again smile if they practice the act before a mirror. We frequently first notice a difference when the patient smiles. The return of emotional expression must not be anticipated. Even in spontaneous recovery, movement on the affected side is not synchronous with that on the sound side. There is also a difference in voluntary and involuntary movements.

Associated movements are recognized as an objection to anastomotic operations. I have never found in my patients that hypoglossal movements were very disturbing. Patients frequently ask which nerve one intends to use. If one says hypoglossal, they may say: "Then half of my tongue will be paralyzed." I say: "Yes, but only one half of the tongue will be affected, and speech and deglutition will not be altered." With the descendens noni, there are no associated movements to embarrass the patient. In discussing the use of a nerve transplant we should remember that bridging of the defect is not new. Dr. Huber, working in governmental laboratories during 1917, 1918 and 1919, tried many types of anastomoses and grafts. In reviewing Huber's original article, I found that the length of his grafts was 3 cm. We hoped after this publication that a panacea for gunshot wounds had been obtained by posture, rerouting the nerve or stretching, but found that for defects larger than from 6 to 10 cm. the situation seemed hopeless and a transplant was needed. Grafts for shotgun and shrapnel wounds were found to be almost hopeless and usually a total failure. In 1919, I used grafts in two cases and secured regeneration, but these were exceptions rather than the rule.

A graft, 3 cm. in length, restored function in the experimental laboratory; in human beings a graft of 10 cm. or more did not restore function. It is therefore not surprising that in the facial canal, with a defect of from 2.5 to 3 cm., results were gratifying. When damage is done during a mastoid operation, one should probably repair the injury by direct suture rather than by a graft.

Dr. William G. Spiller: I presume that in using grafts, experiments have been performed on transferring the nerves of one animal to another, for example, from an ape to a gibbon. It is not advisable to use a nerve from an animal for a graft in man. Sir Charles Ballance is the greatest authority in the world on nerve anastomoses. His first case gained prominence in 1895, and he has carried on his work through all the years since then. He has, however, surpassed himself recently by grafting a sympathetic nerve to the peripheral facial stump. He reported that in the ninth month in one of his animals the face was symmetrical, and at the eleventh month, by exposing the brain and applying an electrode to the appropriate area, muscular contractions were obtained in the facial distribution. One can understand how it is possible to get motor function with the glossopharyngeal and spinal accessory nerves, but in dealing with the sympathetic nerves one has no evidence of their representation in the brain cortex. Voluntary movements have not been demonstrated to be absolutely normal in the animals operated on. Sir Charles Ballance once asked me what the condition of the cerebral cortical cells in the region of Rolando would be after the facial nerve was cut so as to destroy from 6 to 12 mm. of its substance. This would depend on the age of the patient. When a nerve is destroyed in a young animal, the cells in the cortex representing those nerves may disappear; in older animals, the cortical cells may survive. Cells of the first neuron are probably not damaged when the cells of the second neuron are destroyed.

Dr. B. Alexander Randall: In a case that I have mentioned several times the patient had facial neuritis following a mastoid operation. There was stiffening

of the face and increasing loss of power but no paralysis. The condition disappeared after many months, but some residual symptoms were noted three years after operation. Mere digital manipulation and massage (after the neurologic department thought that there was no possibility of recovery) brought about improvement. Later, function was again lost, with the appearance of middle ear infection, and spasticity became permanent. Successful results may occur temporarily, but some of them may not be permanent.

DR. JAMES A. BABBITT: I asked Dr. Duel what had been developed of a practical nature in these research studies. The substitution of this new method seems to provide results without the associated movements. The technic is practical, but seems to be essentially that of the skilful neurosurgeon.

DR. ROBERT J. HUNTER: Facial palsy sometimes precedes the mastoid operation, and after operation many of these patients recover. Could Dr. Duel's and Sir Charles Ballance's method be applied to such cases?

DR. ABRAHAM ORNSTEEN: I note that in two of Dr. Duel's cases there were different results. The woman mentioned had had paralysis for a year before operation was attempted. An infant was operated on almost immediately after injury. The time element therefore seems important. In the adult, there is little recovery in the lower part of the face. The animals were operated on shortly after dehiscence had occurred, and conditions were therefore favorable. Has Dr. Duel seen favorable results in animals when the injury has been of long duration? One cannot expect good results when there is a long peripheral stump. In order to secure good results in traumatic mastoid cases, this operation should be done as early as possible.

DR. ARTHUR B. DUEL: The point raised by Dr. Ornsteen may be answered by a point brought out by Dr. Frazier. He spoke of the premonition that a patient has of a sense of power in the face. In our animals, we traumatized the nerve and there was no opportunity for degeneration of muscle, for the reparative process began at once following the introduction of the graft. In the infant whose case I reported, repair was made at once, and although there was the reaction of degeneration, recovery was about as rapid as in Bell's palsy. The longer the delay before repair, the greater is the degeneration of the fiber and muscle, and the less favorable the prognosis.

When the response to galvanic electricity occurs in the muscle, repair of the dehiscence will be followed by recovery, but when the response is poor to galvanic currents, the prognosis is poor. Two adults mentioned the sensation that Dr. Frazier has described. It is the certainty of something about to happen in the involved area. Trophic influences appear before the muscle actually functions. The reason why apparently perfect recovery occurs in animals while there is less perfect recovery in human beings has been given by Hunt, who described different levels from which stimulation comes: paleokinetic influences, which spring from lower levels and have to do with essential movements such as those of the face in eating, and neokinetic influences, springing from higher levels and dealing with the expression of emotions (response to music, speech, etc.). Neokinetic influences are probably present only in human beings.

As to the question of the time of operation on a nerve after injury, Sir Charles Ballance has advised: "Do it now; do not wait." Poor results may be obtained by instituting medical treatment before trying operation. In trauma there is pressure on a nerve. Therefore, uncover it so that permanent loss of function may be prevented. With proper technic, one will not do as much permanent injury to a nerve as by waiting.

Book Reviews

Intracranial Pyogenic Diseases. A Pathological and Clinical Study of the Pathway of Infection from the Face, the Nasal and Paranasal Air-Cavities. By L. Logan Turner and F. Esmond Reynolds. Price, 12 shillings, 6 pence, net. Pp. 271, with 82 illustrations. Edinburgh: Oliver & Boyd, Ltd., 1931.

This is a volume of 271 pages with 82 illustrations, 21 in colors. It is a compilation of an investigation extending over nine years into the pathways of infection to the brain and its membrane from primary septic foci on the face and in the nasal and paranasal air cavities. All of the illustrations have been made from the authors' own preparations. The main object of the investigation was to study as completely as possible the pathways of pyogenic infection to the brain and meninges from primary septic foci and in the nasal cavities. The observations and conclusions are based on the examination of 53 cases of pyogenic infection in which subsequent complications developed. The primary foci were located as follows: in 4 cases, on the face; in 42 cases, in the paranasal air cavities; in 1 case, in the fauces, and in 5 cases, in the middle ear. Excluding the 5 cases of aural origin, the complications arose from the paranasal air cavities in 90 per cent of the series. This comes rather as a surprise, since our own experiences have led us to expect a much larger percentage from infection of the middle ear. An outstanding fact is the large proportion of these intracranial complications which followed in the wake of operations on infected paranasal sinuses.

In our own experience we have seen a number of cases presenting intracranial complications from infection in the paranasal sinuses, but have not had a single experience in which an intracranial complication followed as a sequel to an operation on the nasal sinuses. Is this difference in clinical experience to be found in a difference in the virulence of these septic infections as found in Edinburgh and here, or is it that we have perhaps been more prone to defer operative intervention until a complication has become apparent? Or is the difference in clinical experience to be found in the difference in operative technic? We are usually satisfied to secure ample drainage for the infected sinuses. Is the tendency in Edinburgh to aim rather at a more radical effort to eradicate the infected focus?

This research is a model for the investigation of a difficult clinical problem, and we recognize here a contribution that will constitute one of the medical classics.

Directory of Otolaryngology Societies *

NATIONAL

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Gabriel Tucker, Bronchoscopic Clinic, Hospital of the University of Pennsylvania, Philadelphia.
Secretary: Dr. John J. Shea, 1018 Madison Ave., Memphis, Tenn.
Place: New Orleans. Time: May 9-13, 1932.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. S. Hanford McKee, 1528 Crescent St., Montreal, Canada.
President-Elect: Dr. B. R. Shurley, 62 W. Adams Ave., Detroit.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: Montreal, Canada. Time: Sept. 19, 1932.

American Bronchoscopic Society

President: Dr. Louis H. Clerf, 128 S. Tenth St., Philadelphia.
Secretary: Dr. Richmond McKinney, 899 Madison Ave., Memphis, Tenn.

American Laryngological Association

President: Dr. George E. Shambaugh, 122 S. Michigan Ave., Chicago.
Secretary: Dr. George M. Coates, 1721 Pine St., Philadelphia.

American Laryngological, Rhinological and Otological Society

President: Dr. Max A. Goldstein, 3858 Westminster Pl., St. Louis.
Secretary: Dr. R. L. Loughran, 33 E. Sixty-Third St., New York.

American Otological Society

President: Dr. L. W. Dean, Washington University, St. Louis.
Vice President: George L. Tobey, 270 Commonwealth Ave., Boston.
Secretary: Dr. Thomas J. Harris, 104 E. Fortieth St., New York.

New Zealand British Medical Association, Ear, Nose and Throat Section

Chairman: Dr. James Hardie Neil, Auckland.
Secretary: Dr. W. Gillies Borrie, Auckland.
Time: March 28, 1932.

SECTIONAL

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. F. M. Sulzman, 1831 Fifth Ave., Troy.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

New England Otological and Laryngological Society

President: Dr. Harry P. Cahill, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Lyman G. Richards, 319 Longwood Ave., Boston.

Pacific Coast Oto-Ophthalmological Society

President: Dr. W. F. Hoffman, 317 Summit Ave., Seattle.
Secretary-Treasurer: Dr. Frank Friesen, 727 W. Seventh St., Los Angeles.
Place: Seattle: Time: June 30-July 2, 1932.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. Albert E. Hillis, 1115 Pacific Ave., Tacoma, Wash.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Sioux Valley Eye and Ear Academy

President: Dr. John B. Gregg, 101 S. Main Ave., Sioux Falls, S. D.
Secretary-Treasurer: Dr. Frederick H. Roost, 420 Trimble Bldg., Sioux City, Iowa.
Place: Sioux City, Iowa. Time: November, 1932.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Wiley R. Buffington, 211 Camp St., New Orleans.
Secretary: Dr. W. D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: Birmingham, Ala. Time: November, 1932.

Southwestern Michigan Triological Society

President: Dr. C. W. Ellis, 215 N. Walnut St., Lansing.
Secretary: Dr. H. B. Weinburgh, 301 Seymour St., Lansing.
Time: Third Thursday of alternate months.

STATE

Colorado Oto-Laryngological Society

President: Dr. F. J. Peirce, 511 N. Main St., Pueblo.
Secretary: Dr. Rex L. Murphy, Metropolitan Bldg., Denver.
Place: Assembly Room of Metropolitan Bldg. Time: First Saturday of each month from October to May.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. M. H. Merriman, 115 Prospect St., Waterbury.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. William L. McDougall, 50 Whitehall St., S. W., Atlanta.
Secretary-Treasurer: Dr. William O. Martin, Jr., 412 Medical Arts Bldg., Atlanta.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. R. Gillum, 221 S. Sixth St., Terre Haute.
Secretary: Dr. J. Kent Leasure, 611 Hume-Mansur Bldg., Indianapolis.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. Gordon F. Harkness, 215 Main St., Davenport.
Secretary-Treasurer: Dr. W. F. Boiler, 105 East Iowa Ave., Iowa City.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Wilfred Haughey, 303 Post Bldg., Battle Creek.
Secretary: Dr. H. O. Westervelt, 239 Pipestone St., Benton Harbor.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Douglas Wood, 74 S. Ninth St., Minneapolis.
Secretary-Treasurer: Dr. W. E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month, October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. L. H. Huber, Livingston.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.
Time: Semi-annually.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. R. T. Atkins, 4 W. 53d St., New York.
Secretary: Dr. D. F. Gillette, 109 S. Warren St., Syracuse.
Place: Buffalo. Time: May 23-24, 1932.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. F. L. Wicks, Valley City.
Secretary-Treasurer: Dr. W. L. Diven, National Bank Bldg., Bismarck.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. H. M. Hendershott, 193 Eleventh St., Portland.
Secretary: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Frank W. Dimmitt, 195 Thayer St., Providence.
Secretary: Dr. N. A. Bolotow, 108 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. D. Jones, 3116 Live Oak St., Dallas.
Secretary: Dr. A. F. Clark, 1034 Nix Bldg., San Antonio.
Place: Fort Worth. Time: December, 1932.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. F. M. McHugh, 17 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. W. Leroy Smith, Erza Thompson Bldg., Salt Lake City.

VIRGINIA SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. C. P. Jones, 3117 West Ave., Newport News.
Secretary-Treasurer: Dr. Henry Grant Preston, Harrisonburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. G. A. Smith, Montgomery.
Secretary: Dr. O. H. Bobbitt, Medical Arts Bldg., Charleston.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Charles W. Buvinger, 50 Washington St., East Orange.
Secretary: Dr. Earl LeRoy Wood, 192 Roseville Ave., Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

ATLANTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. L. McDougall, 50 Whitehall St., S.W., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., 412 Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

BALTIMORE CITY MEDICAL SOCIETY, SECTION ON OTOLOGY AND LARYNGOLOGY

President: Dr. James J. Chisolm, Medical Arts Bldg., Baltimore.
Secretary: Dr. Louis Diener, 2449 Eutaw Pl., Baltimore.
Place: 1211 Cathedral St. Time: 8:30 p. m., last Friday of each month.

Buffalo Oto-Laryngological Society

President: Dr. John F. Healey, 925 Delaware Ave., Buffalo.
Secretary-Treasurer: Dr. Herrmann E. Bozer, 468 Delaware Ave., Buffalo.
Place: Buffalo Club. Time: Second Tuesday of each month from September to May, inclusive.

Chicago Laryngological and Otological Society

President: Dr. Howard C. Ballenger, 25 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Austin A. Hayden, 25 E. Washington St., Chicago.
Place: Medical and Dental Arts Bldg., 185 N. Wabash Ave. Time: 6 p. m., first Monday of each month from October to May.

Cincinnati Oto-Laryngological Society

President: Dr. M. F. McCarthy, Union Central Bldg., Cincinnati.
President-Elect: Dr. Henry M. Goodyear, 19 Garfield Pl., Cincinnati.
Secretary-Treasurer: Dr. Samuel Seltz, Provident Bank Bldg., Cincinnati.
Place: University Club. Time: 6:45 p. m., second Tuesday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. M. W. Jacoby, 2323 Prospect Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 1065 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.
President: Dr. Edward D. King, 707 Race St., Cincinnati.

College of Physicians of Philadelphia, Section on Ophthalmology

Chairman: Dr. Ralph Butler, 2007 Chestnut St., Philadelphia.
Clerk: Dr. Karl M. Houser, 1826 Pine St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., third Wednesday of each month from October to June.

Columbus Ophthalmological and Otolaryngological Society

Chairman: Dr. Ivor G. Clark, 188 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Francis W. Thomas, 327 E. State St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Otolaryngology

President: Dr. L. M. Sellers, 1717 Pacific Ave., Dallas, Texas.
Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. Cecil C. Jones, 1121 Equitable Bldg., Des Moines, Iowa.
Secretary-Treasurer: Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: Third Monday of each month, September to May, inclusive.

Detroit Oto-Laryngological Society

President: Dr. Raymond Goux, 1551 Woodward Ave., Detroit.
Secretary-Treasurer: Dr. R. Lee Laird, 513 David Whitney Bldg., Detroit.
Place: Maccabees Bldg. Time: Third Wednesday evening of each month from October to May.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. R. H. Needham, 1304 Medical Arts Bldg., Fort Worth, Texas.
Secretary: Dr. Van D. Rathgeber, 1011 Medical Arts Bldg., Fort Worth, Texas.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Carl F. Snapp, Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Walter W. Oliver, 26 Sheldon Ave., Grand Rapids, Mich.
Place: Various local hospitals. Time: 6:30 p. m., third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. L. W. Raney, 609 Fannin St., Houston, Texas.
Secretary: Dr. J. F. Gamble, 201 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. B. J. Larkin, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Otolaryngology

President: Dr. W. E. Keith, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. O. S. Gilliland, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. R. R. Montgomery, 102 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Russell T. Uhls, 823 Security Bldg., Long Beach, Calif.
Place: Pacific Coast Club. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section of Ophthalmology and Otolaryngology

President: Dr. Eugene R. Lewis, 727 W. 7th St., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 727 W. 7th St., Los Angeles.
Place: California Hospital, assembly room. Time: 8 p. m., first Monday of each month, May to November, inclusive.

Medical Society of the County of Kings, Section on Otolaryngology

Chairman: Dr. Edward Leo Berger, 903 Park Pl., Brooklyn.
Secretary-Treasurer: Dr. Oscar Rodin, 893 Eastern Parkway, Brooklyn.
Place: Library Bldg., County Society, 1313 Bedford St., Brooklyn.
Time: 9 p. m., second Wednesday of January, March, May and November.

Medical Society of the District of Columbia, Section on Ophthalmology and Oto-Laryngology

Chairman: Dr. James A. Flynn, 1511 Rhode Island Ave., Washington.
Secretary: Dr. E. R. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

The Milwaukee Oto-Ophthalmic Society

President: Dr. T. F. McCormick, 324 E. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Hilliard Wood, 700 Church St., Nashville.
Secretary-Treasurer: Dr. H. C. Smith, Medical Bldg., Nashville.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month, October to June.

New Orleans Ophthalmological and Otological Society

President: Dr. W. R. Buffington, 211 Camp St., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: January to June and October to January—third Thursday of each month.

New York Academy of Medicine, Section of Laryngology and Rhinology

Chairman: Dr. John M. Loré, 240 West End Ave., New York.
Secretary: Dr. Robert E. Buckley, 2 E. 103d St., New York.
Place: 103d St., and 5th Ave. Time: 8:30 p. m., fourth Wednesday of each month from October to May.

New York Academy of Medicine, Section of Otology

Chairman: Dr. Clarence H. Smith, 2 E. 54th St., New York.
Secretary: Dr. James Garfield Dwyer, 57 W. 57th St., New York.
Place: 103d St. and 5th Ave. Time: Second Friday of each month from October to May.

Omaha and Council Bluffs Ophthalmological and Otolaryngological Society

President: Dr. W. P. Haney, 107 S. 17th St., Omaha.
Vice President: Dr. L. G. Howard, 532, 1st Ave., Council Bluffs, Iowa.
Secretary-Treasurer: Dr. W. A. Cassidy, 1620 Medical Arts Bldg., Omaha.
Place: University Club, 1914 Harney St., Omaha. Time: Dinner, 6 p. m. Program, 7 p. m., third Wednesday of each month from October to May.

Philadelphia Laryngological Society

President: Dr. Arthur J. Wagers, 1429 Spruce St., Philadelphia.
Secretary: Dr. Herman B. Cohen, 1301 Spruce St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., first Tuesday of each month from October to June.

Pittsburgh Otological Society

Chairman: Dr. James Homer McCready, 121 University Pl., Pittsburgh.
Secretary-Treasurer: Dr. Roy C. Cooper, 510 S. Aiken Ave., Pittsburgh.

Richmond Ophthalmological and Oto-laryngological Society

President: Dr. Emanuel U. Wallerstein, Professional Bldg., Richmond, Va.
Secretary: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. E. S. Amsler, 53 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. E. W. O'Brien, 5 N. Goodman St., Rochester, N. Y.
Place: Rochester Medical Association, 13 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ear, Nose and Throat Club

Acting President: Senior member of the executive committee: Dr. Arthur W. Proetz, 3720 Washington Blvd., St. Louis.
Secretary: Dr. B. J. McMahon, 813 Missouri Bldg., St. Louis.
Place: University Club Bldg. Time: Third Wednesday in February, April, October and December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Scott Applewhite, 706 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Wilber F. Swett, 490 Post St., San Francisco.
Secretary: Dr. Robert Carson Martin, 384 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Third Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. Claude L. La Rue, Highland Clinic, Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 505 Medical Arts Bldg., Sheveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of each month from October to June.

Spokane Academy of Ophthalmology and Otolaryngology

President: Dr. Paul A. Remington, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. Louis A. Parsell, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. M. G. Brown, 713 Genesee St., Syracuse, N. Y.
Secretary-Treasurer: Dr. A. Harry Rubenstein, Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Tulsa Academy of Ophthalmology and Otolaryngology

President: Dr. Roy W. Dunlap, 108 W. 6th St., Tulsa, Okla.
Secretary-Treasurer: Dr. Marvin D. Henley, 108 W. 6th St., Tulsa, Okla.
Place: Medical Arts Bldg. Time: Monthly.

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